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ANGINA PECTORIS AND ITS MANAGEMENT*

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This subject has been chosen because angina pectoris belongs in a group of the most important diseases, judging from the standpoint of the mortality rate in this country; namely, the cardiovascular-renal. More than three percent of all men drafted in the late war were rejected because of organic heart disease[1]; and in the age groups over 45, organic heart disease causes more deaths than any other one disease[2]. Halsey and others estimate that sufferers from heart disease require ten percent of the total capacity of our general hospitals and constitute more than twenty percent of the visitors to the ambulatory clinics[3].

It is a well recognized fact that cardiovascular-renal diseases demand the attention of every practitioner, with careful study of their prophylaxis, their treatment, and their mortality rate. There should be organized effort, as well as universal individual effort, to study this problem from all angles, so that the frequency of cardiovascular-renal diseases may be diminished.

Angina pectoris is not a new disease, but one which has been known for centuries. A classical description was given by Wm. Heberden, about 1768, and it is almost equal to any that can be given at the present time. Before this period the disease was known by various names, perhaps most often as "asthma convulsivum". Today, angina pectoris has become very common; in all probability more so in this country than in any other part of the world, even England not excepted.

Since the publication of Heberden's description, attempts to explain the whys and wherefores of the symptoms of angina pectoris have been made by the leaders in medicine in every country. Huchard collected all the theories and explanations that had been advanced up to his time and found about eighty, and, no doubt, several have been suggested since then.

The various explanations have usually hinged on the question of whether the cause of the pain and the disease as a whole was due to changes in the first portion of the aorta, the coronary arteries and the heart muscle, or whether it was a type of spasm originating in the nervous system, in which the sympathetic or autonomic systems play a part. More recently the theory has been advanced that the pain is due to increased intragastric pressure, basing this assumption on the fact that a large percentage of the persons having angina pectoris suffer with gastric and intestinal flatulence and experience relief by belching. Verdon has

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written a volume of several hundred pages in which he explains anginal pains by increased intragastic pressure.

At the present time, the best authorities consider one or the other of two definite conditions as the cause of the symptoms of angina pectoris. One theory, sponsored by Sir Clifford Albutt, is that the disease is aortic in origin and involves principally the very beginning of the aorta. The other, advanced by Sir James MacKenzie, explains the pain as myocardial in origin, due to an insufficiency of the heart muscle and relative ischemia because of disease of the coronaries. Vasomotor variations have been held by different observers to be an explanation of certain types of angina. Nothnagel described an "angina vasomotoria".

ETIOLOGY

In spite of all the theories that have been advanced to explain the occurrence of angina pectoris, as the principal etiological factor, we must recognize arteriosclerosis, which may be general or localized. It is important to bear in mind that a patient with angina pectoris may have a perfectly normal blood pressure and no apparent changes in the peripheral arteries and still have a marked sclerosis of the beginning of the aorta and the coronaries. In my last case coming to autopsy, the patient had had a blood pressure of 120 systolic, 80 diastolic, and by palpation no evidence of arteriosclerosis could be noted in any of the peripheral arteries. Yet we found plaques of sclerosis in the arch of the aorta and the coronaries were markedly sclerotic, one being almost occluded.

Age: Hugh Stewart reports the case of a boy of 7 with angina pectoris symptoms associated with aortic valvular disease of rheumatic origin[4], and Albutt[5] describes one following rheumatic fever where the patient was only twelve and a half. In the young, local changes in the coronaries seem the most plausible explanation of the angina symptoms.

Angina pectoris due to syphilis is often found comparatively early in life, generally under 50. But when we consider all cases, the largest group is between 55 and 65, with gradually decreasing frequencies in either direction.

Heredity: With nearly every arteriosclerotic patient we are able to get a history of cardiovascular-renal disease or some constitutional disturbance, such as gout or gouty arthritis, migraine, asthma or obesity. Such persons practically always have an increased amount of uric acid in the blood; and even if in many cases we fail to find any evidence whatsoever of renal inefficiency, I am inclined to think that further study will justify us in believing that these individuals have a constitutionally defective metabolism; or, in other words, an inability properly to care for the protein part of their food. It is likely that their ability to take care of endogenous uric acid is definitely disturbed.

Infection: Both acute and chronic infections play a part in causing early appearance of angina. Lues must be recognized as extremely important, because of the profound changes in the aorta due to syphilis; and the same changes extend into the coronaries. Rheumatic infections must also be considered, since it is a well established fact that lesions of the aortic valve are frequently the result of acute rheumatism. Chronic foci of infection, such as those which occur in teeth, tonsils, gallbladder, and other organs, play a similar role. Our recent epidemics of influenza have undoubtedly hastened the onset of angina pectoris in many individuals.

All these infections are apt to produce what has been described by some as toxic angina; and, in the cases where chronic foci of infection are a cause, great relief is often obtained by their removal. Where degenerative changes have taken place, as in syphilis, and the involvement of the beginning portion of the aorta by rheumatic infections, treatment is not so productive of relief. As closely allied to these etiological factors, we

may include overindulgence in food, tobacco, alcohol, tea, coffee, and the like.

Contributory causes: Any condition causing undue cardiac labor, such as chronic renal disease or the excessive physical exertion of the athlete or the day laborer, must be looked upon as a factor conducive to arterial degeneration.

PATHOLOGY

The pathology of angina pectoris is limited almost entirely to general or localized arteriosclerosis, but the essential features are the degenerative changes in the ascending portion of the aorta and in the coronaries. In a personal interview with Wenckebach, of Vienna, we learned that in a careful study of the aorta in a large number of cases of angina pectoris the most marked pathologic findings were in the first inch of the aorta, the changes noted there frequently extending into the coronaries. In one of my cases extensive arteritis had developed in the beginning of the aorta and the coronaries without any changes in the blood vessels anywhere else in the body.

Associated with the changes in the aorta we nearly always have evidences of chronic myocarditis, or degeneration of the heart muscle. In a large proportion of cases this is probably due to ischemia resulting from partial occlusion of the coronaries.

Recent ventures in the surgical treatment of angina pectoris no doubt will stimulate more careful investigation of the pathologic conditions in the beginning aorta and the coronaries and the relation of these changes to the sympathetic nervous system.

SYMPTOMS

Symptoms of angina pectoris may appear suddenly or develop insidiously. It is important to get a careful history of each case in order to learn of the earliest manifestations. Superficially it would very often seem that the angina came on suddenly, usually after some vigorous exertion or extreme emotional disturbance, and perhaps shortly after a hearty meal. If we inquire in detail we shall find that very frequently the earliest symptoms were manifested in the form of a shortness of breath which came on more easily after moderate exertion than had been the case earlier in life. We also learn that the patient experiences a sensation of tightness or pressure beneath the sternum in conjunction with exertion or emotional stress. These symptoms may have existed for a long time and yet have been given no consideration at all. The patient's blood pressure may have been high for a number of years.

The location of the pain varies in different individuals, but the majority mention a feeling of pressure beneath the sternum, followed by intense pain that nearly always radiates upward towards the neck, the left shoulder, and extends into the arm as far as the wrist. Sometimes a sense of numbness appears first about the wrist or the elbow, later followed by the pain just described. Radiation to the right arm also occurs and occasionally we see a case where both arms become involved. The radiation sometimes extends into neck, jaw, roof of the mouth, or left ear. I have now under observation a case in which the pain begins beneath the left shoulder blade and extends through to the anterior surface of the chest and up into the arms.

When these patients describe their sensations they nearly always volunteer the information that when they stop and rest for a few moments the pain disappears. Others experience relief by belching, and these often insist that the cause of their trouble is gastric in origin.

The philosophy explaining the occurrence of the pain is still rather vague. Jonnesco[6], who was the first to treat a case of angina pectoris surgically, believed that the pain and the other symptoms of angina were due to irritation of a cardio-aortic plexus, describing them as a neuralgia of this region. Vaquez and others lean definitely towards this view. Everyone agrees that an aortitis of more or less intensity exists in all cases of true

angina, and the supposition is that this aortitis irritates the nerve filaments of the cardio-aortic plexus. Because of the occurrence of pain the supposition is that the nerve fibers in this region are sensory in type, but proof of this has not been absolute. Whether or not involvement of the coronaries alone can produce the same symptoms as the classical angina with the aortitis has not been demonstrated.

All cases of chronic aortitis do not develop symptoms of angina. Consequently, it is important for us to distinguish between this condition and angina; as a rule, we find that the aortitis manifests itself by a sense of distress beneath the sternum that is more or less continuous and may be aggravated by exertion. There is usually a definite tenderness on pressure along or near the sternum. In many cases of aortitis the patient is constantly conscious of discomfort, usually described as an ache, rather boring in character, located beneath the sternum. This is not relieved by rest or nitrites.

The pain of aneurysm must also be considered. A recent case under observation developed intense pain beneath the sternum on even moderate exertion, and with it the pallor, perspiration and other symptoms that are usually attributed to angina. Rest and nitrites did not give relief, and the attacks of pain lasted for hours unless an opiate was used. The blood pressure was only slightly increased. The pulse rate was not materially disturbed. An x-ray examination of the aortic arch revealed a fusiform dilatation of the descending portion of the aorta, which explained the occurrence of the attacks of pain.

Among the comparatively young we find a rather large group of individuals who complain of a pain in the region of the heart that as a rule does not radiate and is more or less continuous. There is rarely any sternal tenderness and frequently no evidence of organic changes in either heart or blood vessels. This type of case became especially familiar during the late war and has been described under the term irritable heart, or circulatory myasthenia. It must not be classed as angina and must be treated very differently.

In angina pectoris the blood pressure may be normal or decidedly increased, depending on the presence of renal complications or a marked general arteriosclerosis. Sometimes nervous tension and temperament must also be considered as a source of cardiac embarrassment. My experience is that those with normal or low blood pressure are usually the hardest to relieve, because we often find marked sclerosis of the peripheral arteries with extensive changes in the aorta, resulting in considerable dilatation of the arch. Diastolic pressure is many times found to be low because of this dilatation.

A study of the blood pressure is very essential, as it gives some idea of the burden the heart has to carry. The pulse pressure tells whether or not the myocardial efficiency is being encroached upon, even when at rest. A small pulse pressure should always be considered suspicious. Symptoms of myocardial inefficiency should always be carefully looked for. The capacity of the heart muscle is frequently reduced because of the age of the patient, and often much aggravated by coronary disease; sometimes in addition to this we find a valve lesion, especially aortic regurgitation, that increases the demand on the heart muscle. In angina pectoris the muscular capacity of the heart must be carefully considered because that is the basis for determining the regimen to be prescribed, including exercise and medical therapy.

It is important to ascertain whether or not shortness of breath comes on with various forms of exercise. It is not sufficient just to ask the patient whether he is short of breath, but one should inquire as to certain definite acts of exertion, such as climbing stairs or going up a slight incline, to find out when discomfort is felt. Often a history of gradually increasing dyspnea on exertion will be apparent.

The patient will also tell you that the pulse rate has increased during this period in which myocardial failure has been developing. The rate will frequently be found to be above 80, and unduly increased by slight exertion. Occasionally we get a pulse rate that drops decidedly below normal, and in such cases we often learn that the patient notes an irregularity in the pulse on exertion. The irregularity is most apt to appear before or during a paroxysm of pain.

It has not seemed advisable to attempt to give any description of the paroxysm, in its many variations; but all types of vasomotor disturbance, with dyspnea, pallor and perspiration, are present in all gradations, according to the intensity of the paroxysm.

GENERAL MANAGEMENT

The management of a case of angina pectoris is an important problem for general practitioners because each will some time have the responsibility. The psychology of every patient must be thoroughly understood. The individual who is apprehensive and easily alarmed must be dealt with in the most optimistic manner in order to dissipate fear and worry. Many times it is essential not to use the term angina to a patient, calling it instead a disease of the aorta that nearly everyone has after the age of fifty. Optimism with all patients is very essential. Hope must be maintained.

Exercise: Base the kind and the amount of exercise on the length of history and the occurrence of dyspnea and on the patient's apparent capacity to move about without the production of pain. Insist that they rest a certain period after meals and that whenever they start to walk or take any form of exercise it must be done slowly and gradually. One may progressively increase the effort permitted, provided the myocardium grows in capacity. Vigorous effort or emotional disturbance increases the rate and the vigor of the cardiac contractions, and the stretching of the aortic ring in all probability accounts for the occurrence of the pain. Myocardial fatigue may also be a factor.

In the severe cases, and especially after a very intense paroxysm, many persons are best treated by a period of complete rest, both physical and mental, with freedom from anxiety and worry. Long periods of rest have never in my experience proved sufficiently beneficial to warrant such a program for the angina alone. If theire is marked myocardial decompensation, then benefit is obtained. The daily routine for the average case should be nine or ten hours in bed at night, with periods of rest and relaxation during the day, particularly after meals, or after the usual duties of the day have been performed.

Diet: The diet must always be simple, and it should be varied according to the extent of the general arteriosclerosis, the renal involvement and the gastro-intestinal conditions. The patient who complains of a great deal of flatulence and is relieved by belching is best served by small meals and the avoidance of fluids of all kinds with meals. Repeated examinations of the gastric contents of this group of patients practically always revealed a normal gastric juice, so that the use of alkalies or acids is not ordinarily indicated. Carminatives may be of value in inducing belching.

The individual with renal disease or marked hypertension should have a low protein diet, basic in character, as outlined by Sansum[7] and Blatherwick. Fruits and other laxative foods should be taken freely in order to keep the bowels active, instead of using salines or purgatives. A low salt intake is in order in cases of renal involvement or marked arterial hypertension. A salt-free diet, however, in my experience has not been of any value excepting where there is edema.

PHYSIOTHERAPY

In nearly all cases mild hydrotherapeutic measures are of value. The purpose is to equalize the circulation, and special effort should be made to increase the circulation of

the skin, thus relieving internal congestion and somewhat lightening the work of the heart. In many cases this must be undertaken slowly, beginning with dry friction to the skin, later using cold towel rubs, and finally salt rubs and graduated showers or pail pours. Definite instructions must be given regarding the temperature of the water. Very often the shock of the cold can be counteracted by the use of a hot foot bath or fomentations over the abdomen before the cold application. The neutral, the Nauheim, or the simple saline baths have their place when given at proper temperatures and for the right length of time. The object is to equalize the circulation in the extremities. Very often great benefit is derived from the relaxing effect on the nervous system.

In the case of hypertension with renal involvement, sweating procedures may be employed if used progressively. I have often been surprised to find how well many of these patients will tolerate the electric light bath followed by graduated showers, without the production of a paroxysm.

Electrotherapy also has its place, diathermy or autocondensation being a source of great benefit. It tends to relax the patient and often lowers the blood pressure, thus relieving the burden on the heart. Massage is valuable, especially in cases where rest is the chief form of therapy. It is often necessary to begin with massage to the extremities only, carefully increasing it in vigor from time to time. Heavy massage to the abdomen should always be avoided in angina pectoris and in cases of marked arterial hypertension.

MEDICINAL TREATMENT

The object of medicinal treatment is, first, to relieve the spasm when the paroxysm is present; and, second, to improve myocardial inefficiency, and if possible to decrease the aortitis and the involvement of the coronaries. For the paroxysms, the nitrites practically always give relief. Amylnitrite is used by some, nitroglycerin by others. Nitroglycerin, as a rule, is more practical because a definite dose can be given and the relief is usually immediate. I have found 1/150 to 1/200 grain to be sufficient in a large proportion of cases. It is important that the patient be instructed simply to place the tablet under the tongue and not to attempt to swallow it or take water with it. The dose can be repeated in a few minutes if the first has not given relief. We can also advise the patient to take a tablet when about to do something which will probably induce an attack of pain.

Some recommend the use of antispasmodics, as benzol benzoate or its derivatives, while the daily use of atropin is advocated by others. Diuretin has been advised, with the idea that it improves the circulation in the coronaries. Five or more grains in capsules, three or four times a day, sometimes prove beneficial. In cases of myocardial inefficiency where there is shortness of breath or edema the use of digitalis is productive of the greatest relief. In fact, it is safe to give digitalis in nearly every case of angina pectoris, provided moderate doses are used; and my experience has shown it to be the most satisfactory remedy for reducing the frequency of attacks and relieving patients of the symptoms of failing compensation. In nervous or apprehensive individuals the use of hypnotics must be considered. In cases of insomnia or restless sleep, the moderate use of bromides, or broken doses of veronal, will often induce sound sleep and be of great benefit to the patient. In cases where syphilis is a factor, iodides, mercury and salvarsan should be used, depending somewhat on the symptoms of the case. In the advanced cases, salvarsan should be used with extreme caution and initial dose should be not more than one-tenth or two-tenths of a gram. If the iodides are used, they should be given in fairly vigorous doses over a long period of time.

SURGICAL TREATMENT

We have reached the point where surgical treatment of angina pectoris

is to be considered. Jonnesco[6] is the pioneer, and if this form of treatment proves satisfactory his name should be famous for it alone. Coffy and Brown[9], of San Francisco, and Holmes and Shackleton, of Chicago, are the pioneers in this country. Eppinger, in Wenckebach's clinic, has also treated cases surgically.

The severing of the superior sympathetic ganglion on the left side is the operation of Coffy and Brown[9], Holmes and Shackleton. Others certainly will try this form of treatment and the reason for being optimistic as to the outcome is the fact that practically every patient who has been operated upon has been relieved of the angina pains. The last report of Jonnesco[8] was that in his first case relief has continued over a period of five years. Every practitioner should watch with keen interest the development of a technic which will make generally available an efficient surgical treatment of a disease so widespread and important as angina pectoris.

SUMMARY

1. Angina pectoris is of such frequent incidence that it of necessity becomes the problem of each general practitioner.

2. Two theories as to the cause of the disease are now considered; first, the disease is aortic in origin and involves mainly the beginning of the aorta; second, the pain is myocardial, due to an insufficiency of the heart muscle and relative ischemia because of disease of the coronaries.

3. Arteriosclerosis is the principal etiological factor. Both acute and chronic infections are important causes, while anything that induces excessive cardiac labor may contribute to arterial degeneration.

4. The essential pathologic features are the degenerative changes in the ascending portion of the aorta and in the coronaries. Associated with these are nearly always evidences of chronic myocarditis.

5. Symptoms of angina pectoris may appear suddenly or be a gradual development.

6. The most likely explanation of the pain is that the nerve fibers involved (the cardio-aortic plexus) are sensory in type.

7. Study of the blood pressure and the myocardial inefficiency is most important.

8. Exercise should be limited and rest periods frequent.

9. A simple diet is demanded, varied according to general arteriosclerosis-renal involvement and gastrointestinal conditions.

10. Physiotherapy, including hydrotherapy and electrotherapy, is a useful remedial agent.

11. Medicinal treatment should seek to relieve the paroxysms, improve myocardial inefficiency and decrease the aortitis. The nitrites, particularly nitroglycerin, are to be used for relief of the paroxysms. Benzol benzoate or its derivatives, atropin, or digitalis, may frequently be employed, and in some cases hypnotics are also indicated. Iodides, mercury and salvarsan, are to be considered in the syphilitic cases of angina.

12. Surgery has been tried in a few cases and the outlook for its successful application to angina pectoris is good.

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ROENTGENOLOGICAL SYNOPSIS OF PULMONARY TUBERCULOSIS WITH SPECIAL REFERENCE TO THE DIAGNOSIS OF CAVITATION*

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The chief value of roentgenology in pulmonary tuberculosis is prognostic rather than diagnostic. Not that the science of roentgenology lacks great diagnostic value, but in this respect it ranks second. We hope to show further on that there are non-tuberculous lesions which may cast shadows similar to those that are truly tuberculous, and because of this and other reasons of only less importance the closest co-operation between the clinician and the roentgenologist is terefore imperative.

Years of closest scrutiny in the field of pulmonary tuberculosis has taught us to believe that roentgen signs in pulmonary tuberculosis appear at times much before physical signs make their appearance, and much after the constitutional behavior of the patient has directed the eye of the clinician towards tuberculosis. To prove these facts, and to deprive our opinion of a personal character, we invite the attention of our colleagues to the well known and generally accepted fact, that roentgenology will invariably show a more extensive lesion than physical signs, for the reason that lesions not yet giving rise to physical signs cast shadows upon the film. And, if this is true of a well established process, why is it not equally true in early lesions? Times we have been approached by keen specialists with the following words: "This patient has tuberculosis; find the lesion". The constitutional symptoms have told the clinician of the tuberculous nature of the case, but the roentgen signs were to him a necessity for the completion of his diagnosis.

Recently a case came under our observation. A young woman with many of the constitutional symptoms of tuberculosis, who had been pronounced tuberculous three years before. At that time physical signs, sputum and x-ray examination were negative. Further x-ray research at this time warranted the diagnosis of chronic cholecystitis and duodenal ulcers. This was confirmed by laparotomy.

Recently Dunham has called attention to the similarity of the constitutional symptoms between sinus infection and tuberculosis.

Activity cannot be diagnosed by the x-ray, neither can the physical signs be relied upon always. One of New Mexico's foremost specialists has been a sufferer from tuberculosis for twenty-five years. He tells us that he has not had a single symptom for the past fifteen years, yet the physical signs are there to bespeak the lesion. How then are the roentgenologist and the clinician to proceed in dealing with tuberculosis? Are they to ignore constitutional symptoms because they are concomitant with other conditions, or physical findings because at times they are absent or persistent, or x-ray studies because their shadows are not characteristic? The clinician and the x-rayist should never disagree; not, if they set aside personalism, and with the interest of the patient at heart, analyze coolly and conscientiously the information at hand. Thus only can they safely travel through the still dark fields of tuberculosis.

If the exactness of the size of a pulmonary lesion, its accurate location, the true condition of the pulmonary parenchyma and the pleura, the degree of pulmonary function in reference to the degree and the character of the infection, the presence of excavation, etc., could be determined in a true fashion by physical examination alone, the science of roentgenology, in so far as pulmonary tuberculosis is concerned, would be dealt a severe blow, for here is where the x-ray examination shines with unique

(*Read before the El Paso County Medical Society, El Paso, Texas.)

brilliancy. Very early, as well as very old lesions, may give indefinite physical signs, yet both are of greatest concern to the clinician; the former in establishing a diagnosis, the latter in determining the extent of the pulmonary involvement and the amount of individual resistance. In advanced tuberculosis, the clinician is surely remiss if he fails to avail himself of the aid of roentgenology. In cavitation, for instance, there is no marked variation in the physical signs from the moment excavation is established until healing takes place and the cavity is walled off. How, then, is he to arrive at prognostic criteria? The same holds true of other phases of pulmonary tuberculous pathology. Again, can he, the clinician, determine by physical examination and in an accurate manner the amount and kind of fibroid proliferation and the stage of the healing process?

A combined skiagraphic and fluoroscopic study is a necessity in certain types of cases, for the study of shadows alone will not determine how the lungs are performing their mechanical and physiological task. Whether or not the diaphragmatic excursion is within normal limits; the extent and the cause of limited pulmonary excursion; the influence that extensive changes in the mesial pleura or marked fibrosis along the descending bronchial trunks may have in circulatory disturbance; and if displacement and even distortion of a thoracic organ is momentary or permanent.

There is no subject that has taken the time, or has occupied the minds of those of us who have chosen roentgenology as our specialty more than the subject of pulmonary pathology, because the shadows cast upon the x-ray film by pulmonary changes are the most difficult to interpret. In order fully to comprehend the meaning of the different shadows, the physician should be well acquainted, first, with the degree of x-ray absorption of a given tissue; second, with the pathological changes possible in the organ to be examined; and last, but not least, with the anatomy and physiology of the organs shown on the skiagram. Gas, fat, muscles, fibroid tissue, bones and fluid, all have different degrees of power of x-ray absorption, according to their atomic weights, gas the least, fluid the most, so that if we could take different layers of equal thickness of the above mentioned elements and an equal x-ray exposure be given, the x-ray plate would reveal a gradual increase in density from the gas to the fluid.

There is no such thing as a typical normal chest radiogram. The normality, however, can be established at the moment a plate is studied, always taking into consideration the age, occupation, degree of chest muscular development and previous pulmonary diseases of the patient. Let us not forget previous pulmonary diseases! Once that the normal shadows have been observed and their meaning understood, then one can conceive the variations from a physiological into a pathological state. No matter what pathological condition we may choose, it is impossible to give an equal description of the same in all cases, for the reason that no two lesions are, histopathologically speaking, alike. Hence, they necessarily cast upon the skiagram different shadows. We will, then, discuss this matter step by step, studying the pathology of pulmonary tuberculosis in its different phases and stages in conjunction with the shadows cast by them in the roentgenograms.

The reading of chest conditions by means of flat plates is a past performance. Flat plates should be used only when and where stereoscopic plates are not feasible. Stereoscopic plates have the great advantage that they indicate the location and the depth of the pulmonary lesion. They will also differentiate between pleural, pulmonary or extrapulmonary conditions. In tuberculosis, especially, they resolve the shadows into their components and thus give a truer idea of the density of the infiltration, and finally, in the average case, they furnish the clue for the roentgenologist to inform the clinician

of the relation between the thoracic wall and a given lesion.

Although pathologists seem to be in full accord as to the pathological changes which take place in pulmonary tuberculosis, there is a lack of unanimity in the classification of the different types. Some will classify the disease according to the supposed mode of infection, while others will base their classification upon the pathological course of the lesion. We feel that in the interpretation of x-ray findings it matters not in which way the organism enters the tissues. So, for the purpose of x-ray study, we will divide pulmonary tuberculosis into four main types: Miliary, chronic ulcerative, chronic fibroid and glandular.

MILIARY TUBERCULOSIS

Once a pathological specimen of this type of pulmonary tuberculosis is studied, there will remain in our minds always the vivid picture of those small grayish, translucent areas, representing the miliary tubercles imbedded in the perivascular connective tissue, scattered in myriads throughout the lung substance. They are to be distinguished from the characteristic conglomeration of tubercles seen elsewhere, not alone by their minute size, but by their irregular configuration. When a fresh cut is made in a miliary lung and the tissues gently squeezed, a certain amount of exudation of a semi-solid consistency is seen oozing out from the margin of the tubercles. What may we expect the x-ray to show? What is there to intercept, to absorb, the rays in addition to the bony structure and the mediastinal organs, the heart included? Only the normal structure of the lungs made up mainly of connective tissue, bronchi and blood vessels, plus the miliary tubercles. Hence, a radiogram in these cases will show numerous minute areas of radiopacity, varying in size and shape, and of a somewhat irregular outline, scattered in between the radiolucent linear markings of the bronchial tree, evenly and symmetrically distributed throughout the fields reaching or not reaching the periphery. In these cases, the pleura is uniformly inflamed, congested, engorged, so that it absorbs evenly throughout both fields a certain amount of radiation and gives to the radiogram a peculiar blurredness similar to that seen in a photographic film when the object photographed moves slightly. This peculiar blurredness constitutes the only roentgen signs that will aid in the differential diagnosis of a miliary pulmonary infection and cases of pneumosiderosis, which likewise show numerous small areas of hyperdensity disseminated throughout the lung parenchyma. In miliary tuberculosis, the costo-phrenic angles are occluded, not by adhesions, but because of the incomplete excursion of the diaphragm, the result of reduced expansion of the lungs.

CHRONIC ULCERATIVE TUBERCULOSIS

The primary lesion consists of scattered tubercles surrounded by a pneumonic exudation in a congested area. In other words, and grossly speaking, we have an area where fluid is accumulated in the tissues. This collection of fluid will intercept radiation and the infiltrated area will cast upon the film shadows of various sizes and densities, depending upon the quantity and physical qualities of the fluid. As the disease advances and caseation takes place, the shadows coalesce and consolidation over small areas is revealed upon the skiagram by radiopaque shadows more or less spherical in shape within a radiolucent area of marked infiltration. Later, either through the blood or the lymphathic channels or else by the discharge of caseous material into the contiguous bronchi and its aspiration into other parts of the lungs, unless the case is non-progressive, the infection is bound to invade the opposite side and the adjoining lobes, and we then see in the gross pathological specimen, in addition to the inflammatory area previously described, numerous isolated tubercles in the adjoining lung parenchyma. When these tubercles are of recent origin, they cast upon the skiagram

soft, faintly radiolucent shadows of blurred, indefinite outline, the last feature being due to a layer of pneumonic exudate surrounding each tubercle. On the other hand, when the tubercles are of long duration, they reveal themselves upon the film as rounded, cleancut, radiopaque shadows; they are no longer surrounded by an area of exudation and their structure contains a certain amount of calcium salts. It is well to emphasize that the correct estimation of the

Fig. 1. Extensive infiltration involving all lobes left lung and upper and middle right, with collapse of right upper and pleurisy with effusion at the lesser fissure. Notice flat fluid level. Scattered areas of rarefaction at the central portion of both fields are indicative of softening and disintegration of the lung tissue with multiple cavitation. Confirmed by autopsy

shadow cast by the tubercle will have an important bearing on the prognostic side of the question.

After the process of infiltration is well established, when the physiological functions are being lost, when there is no longer gaseous alveolar interchange, when there is hindrance to the capillary circulation and there is likelihood of exudative decomposition, breaking down of the lung tissue begins to take place, and the skiagram shows areas of radioparency, irregular in conformation, and usually in the midst of radiopaque zones. This softening and disintegration has been observed by us only in cases far advanced. If, at this stage, reparative changes take place in the form of connective tissue cell proliferation, the areas of radioparency are supplanted by areas of radiopacity which show themselves upon the film in the form of irregularly shaped shadows with little, if any, lung markings, characteristic of diffused fibrosis. If, however, the ulcerative process proceeds, it will eventually lead to a most characteristic lesion of a tuberculous lung, viz., cavity.

THE CAVITY

The roentgen diagnosis of pulmonary cavitation of tuberculous origin may be at times confusing and rarely impossible. Speaking from the standpoint of the roentgenologist, the liter-

Fig. 2. Extensive involvement of all lobes, right side, with well walled off cavity at the lower portion of the upper lobe. Notice the haziness and undefined outline of the internal border by the shadow cast by the capsule. Autopsy findings revealed cavity somewhat triangular in shape and measuring about 5 c.m. from apex to base, surrounded by a zone of indurated lung parenchyma, where the external border of the capsule was lost. There was no fluid present, the even haziness cast upon the plate being due to the rays having been intercepted by the indurated lung tissue anterior and posterior to the cavity. Two small, acute cavities triangular in shape, at a level with sixth interspace behind. Breaking down of the parenchyma at a level with the eighth behind. Confirmed by autopsy.

ature on this subject does not seem to be, generally speaking, based on autopsy findings to the extent that we would like, and which we have found to be of absolute necessity in the proper interpretation of roentgen shadows cast by pulmonary cavitation, inasmuch as other conditions give physical signs similar to those elicited by parenchymal excavations.

For diagnostic purposes we will speak of pulmonary cavitation as acute and chronic. The acute runs a parallel course to, and is concomitant with, an active destructive process.

If we bear in mind the histological elements that enter into the composition of the lung parenchyma, their distribution and the pathological changes that take place in tuberculous parenchymatous pulmonary infection, the following facts must be taken into consideration in dealing with the study of acute cavitation:

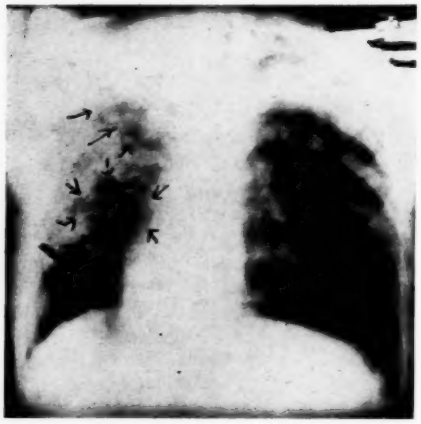

Fig. 3. Extensive infiltration involving the left lung and right upper with moderate infiltration of the middle. Irregular areas of rarefaction at the lower portion of the left upper proved to be at autopsy multiple cavities. Notice large "punched out" area at level with eighth rib behind. Roentgen diagnosis: Cavity. Confirmed by autopsy.

1. That they are improbable in the absence of an extensive involvement.

2. That their shape is triangular, or quasi triangular.

3. That their outline must be irregular.

4. That they show no evidence of capsule.

Cavities are the result of disintegration which is brought about by the pressure of retained secretions within softened portions of the infected lung parenchyma or by liquefaction of caseous areas not in connection with the larger bronchial trunks. Disintegration, therefore, cannot occur in early parenchymatous infiltration. Hence, acute cavitation is not to be expected unless the infection is well established.

Fig. 4. Old infiltration involving the right, upper and middle lobes. Autopsy findings: Induration of upper lobe with cavity formation. Extensive fibrosis.

As to their shape, let us bear in mind the normal distribution of the pulmonary stroma. The tissues that enter into its composition originate in the hilar region, whence they radiate in all directions into the parenchyma in a fan-like manner, forming pyramidal tracings, with the base towards the surface. They surround areas of parenchymatous tissue composed of lobules closely connected to one another by the interlobular areolar tissue. It is an accepted fact that the stromal tissues resist ulceration more than the neighboring parenchyma, therefore, following their histological distribution, acute cavities assume a

pyramidal shape, their internal border being ragged and irregular because of the asymmetry in the course of the destructive process. What we term acute cavities do not show evidence of encapsulation for the reason that they pertain to that type of cases where reparative changes have not taken place. When cavitation takes place, as the result of pressure of retained secretions within disintegrated and softened portions, we see, in serial roentgenograms, a change in the density of the shadow from radiolucency to radiopacity, due to the gradually increasing secretions absorbing the rays. In a majority of the cases the radiopacity changes to radiolucency just as soon as a communication is established between the cavity and the neighboring bronchi, and drainage takes place, because, then, there is no fluid to intercept radiation. If, however, pulmonary cavitation is the result of liquefaction of caseous material, no change in the shadow will take place while the process of cavity formation is going on, because caseated material intercepts radiation at the same rate that secretions do, and unless drainage takes place, a cavity within a caseated area may be overlooked. Once drainage is established, however, an area of radiolucency corresponding to the approximate size of the cavity will be in evidence.

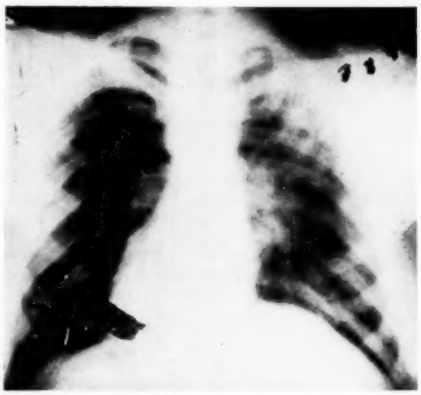

Fig. 5. Extensive fibroid degeneration involving right upper. Physical signs consistent with large cavity. Autopsy findings showed several very small cavities at the upper and diffused infiltration at the middle and lower lobes. The patient died of acute peritonitis of postoperative origin.

A few years ago, and even in the present day, some of our leading authors in pulmonary tuberculosis put forward the assertion that cavities are represented upon the film by areas of radioparency containing no lung markings. For obvious reasons, this is not the case. Acute cavities almost invariably, unless, as it rarely happens, situated in the very periphery, are enveloped by pulmonary tissue which will intercept radiation, not only casting shadows representing lung markings, but also preventing the feature of radioparency.

CHRONIC CAVITIES

We speak of chronic cavities when the process of repair is well advanced and a distinct fibroid wall has been formed around the cavitation. These cavities preserve their original shape, or, at least, have a tendency to triangulation, unless they are of very long standing and are surrounded by a more or less extensive area of induration. Then they attain a circular

Fig. 6. Typical annular shadow, the result of thickened pleura. Notice the size of the ring, which is out of proportion to the amount of infiltration, the cleancut internal border, and the clearness of the enclosed field. No cavity found at autopsy.

shape due to shrinkage of the neighboring tissue. The capsule is represented upon the skiagram by a dense ring-like shadow of varying thickness, having either a well defined or a blurred but rough, ragged, uneven internal border enveloping a radiolucent area. This area may or may not contain lung markings, depending entirely upon the position of the cavity, the thickness of the capsule and the solidity of the surrounding lung tissue. When a cavity is situated deep in the center of the parenchyma, the capsule is thin and the surrounding lung tissue is not indurated, lung markings anterior and posterior to the cavity will cast their shadow within the area of excavation. Per contra, when the capsule is heavy, whether or not the adjoining lung tissue is indurated and regardless of where the cavity is situated, lung markings will not be in evidence, the excavation being covered by a heavy blurred veil of even density and uniform haziness, similar to those shad-

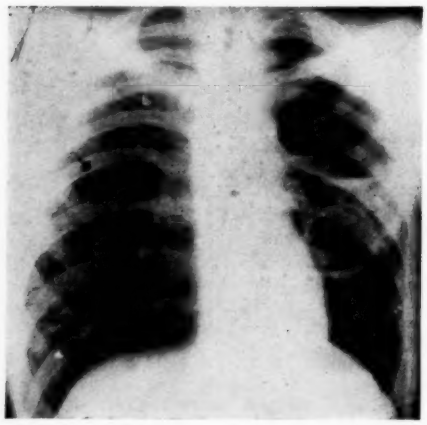

Fig. 7. Two typical annular shadows on the left. Notice in between, pleural shadow with a tendency to annular formation, showing a flat fluid level. Physical signs positive of cavity corresponding to a zone occupied by the superior shadow. Autopsy findings show that physical signs were the result of pleural changes with subsequent cupping of the lung tissue. The middle shadow proved to be a localized pleurisy with effusion, with moderate cupping of the lung parenchyma. (Localized pneumothorax). The inferior shadow was found to be due to annular pleural changes. Notice the infiltration, out of proportion for the size of the annular shadows. On the uppermost portion of the right lower lobe, two small cavities were found. They gave no physical signs, but were diagnosed as such by the x-ray.

Fig. 8. Fibrinous pleurisy throughout the right pleura, with a tendency to circular formation. Notice the sharpness of the internal border in the circular shadow. Intra-lobular pleurisy with effusion at the lesser fissure. The autopsy findings verified the x-ray findings and showed extensive parenchymatous infiltration, with no cavity formation.

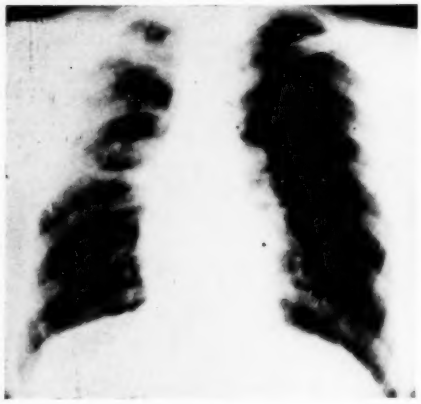

Fig. 9. Well established tuberculous infiltration of all lobes, right side, with pleural changes in annular formation projecting from the hilar region. Autopsy findings showed thickening of mesial pleura and confirmed x-ray findings.

ows cast by markedly thickened pleura, the rays having been absorbed in direct proportion to the thickness and the degree of absorption of the tissue anterior and posterior to the cavity.

DIFFERENTIAL DIAGNOSIS

Four different types of shadows are to be dealt with in the differential diagnosis of tuberculous parenchymal excavation:

1. Those pertaining to true cavities.

2. Those cast by thickened pleura in annular formation.

3. Those cast by localized spontaneous pneumothorax.

4. Those cast by annular shadows of fibroid origin.

Location: Autopsy findings have taught us to believe that cavities, in so far as location is concerned, follow the same lines as an invading tuberculous infection, i. e., they are most commonly found in the uppers, usually just below the clavicle close to the periphery, sometimes at the apex, and seldom at the lower portion of the upper. Cases have been reported with tuberculous cavities at the bases. We have never seen this condition. Pleural shadows may occur anywhere in the field, most commonly posteriorly at or near the central portion. Occasionally, when they are of intralobar origin, they are visualized in the midst of the lung parenchyma; at times they are seen directly behind the front ribs. The type of spontaneous pneumothorax that may be mistaken for cavity is always situated at the apex, unless it accompanies the pleural annular shadows with cupping of the encircled lung tissue when it may occur anywhere, but usually in the central portion of the field close to the posterior surface. Fibroid changes in circular formation are invariably seen originating from the hilar region and are, therefore, in the center of the field, and may travel either forward or backward, sometimes upward and rarely downward.

Shadow Characteristics: The size of a pulmonary excavation varies, but is always in direct proportion to the nature and the extension of the lesion. In those cases following a caseating course from the beginning, cavities usually attain enormous sizes, while in those cases where fibroid proliferation starts early, cavities are small and commonly multiple. Annular shadows of pleural origin may be of any size regardless of the degree of involvement. We have seen pleural shadows that were the only unmistakable evidence of the true nature of the accompanying constitutional condition, the remainder of the field being comparatively clear, showing only scattered shadows to awaken suspicion concerning their real cause. Fibroid shadows in annular formation are to be expected and are met with as a rule in cases of true fibroid phthisis, although, at times, they are seen in the ulcerative type when fibroid changes follow, step by step, the parenchymal infiltration. They are usually small and are seen connected by a stem to the hilar region whence they originate. Spontaneous pneumothoraces of apical origin usually involve the whole of the apex. When associated with pleuritic annular shadows, their size varies considerably. Acute cavities, being deprived of a capsule, or, at least, of a complete capsule, when situated at the very apex, cast a shadow that may be mistaken only with the shadow cast by a spontaneous pneumothorax; in the latter, however, the edge of the collapsed lung can often be made out, and the shadow is one of radioparency containing no lung markings. When acute cavities occur elsewhere, they present a "punched-out" area of radiolucency showing lung markings, of triangular or almost triangular shape, and of irregular, ragged, ribbed outline. There is, to our knowledge, no other pathological entity casting a similar shadow. Chronic cavities, however, no matter where situated, must be differentiated from annular pleural shadows and fibroid ring-like shadows. We have already discussed the nature of the shadow cast by this type of cavity, which is

quite in contrast to those cast by pleural changes in annular formation. Annular pleural shadows have a cleancut, even, smooth internal border much different from the external one, which has a blurred and undefined appearance, the dense shadow gradually fading out as it travels outward away from its axis. On several occasions we have diagnosed triangular shadows showing the above characteristics as of pleural origin, autopsy findings confirming our belief.

Many creditable articles discussing this most deserving subject have appeared recently, and it is astonishing to see groups of men of equally extensive experience holding directly opposite views, all the result of their experience, backed by the force of a well established professional reputation. Between them, with the same force, stand Amberson of the Loomis Sanitarium and J. A. Honeij, with equally feasible ideas as to the pathological significance of pleural changes in annular formation.

We could agree with the recent statement, "pneumothoraces are never annular in contour", if we may add, when accompanied by p l e u r a l changes. Hard knocks and disappointments have taught us to believe in the existence of annular areas of thickened pleura with subsequent cupping of the encircled lung parenchyma. Autopsy findings have placed our error in the limelight by revealing annular pleural changes where findings of a cavity had been foretold because of the leading physical signs and because of what we considered positive x-ray findings. It was true that in the process of separating the adhesions between the parietal and the visceral pleura, much of the structural appearance was destroyed, but there was oftentimes sufficient pathological evidence at hand to prove the existing conditions. On inflation of the lung, a well pronounced umbilication of the parenchyma was observed at the site of the lesion, before inflation was completed, this portion being the last to attain full expansion. After the lungs were properly expanded, they were submerged in water, and in only one case there seemed to be a few air bubbles that did not come up to the water surface, showing that if there were any air leakage, it was almost osmotic in character. This naturally might have been the result of traumatism during the dissecting procedure. The postmortem work performed by us has failed to confirm the theory of ruptured lung as causative of pneumothoraces simulating cavities. Every case of ruptured lung that has come under our observation has been accompanied by an extensive pneumothorax with partial or complete collapse of all lobes of the affected side. When shadows cast by pulmonary excavations, pleural changes, spontaneous pneumothoraces or ring-like fibroid degeneration show the characteristics above described, we have had little difficulty in differentiating them. However, there are a large percentage of cases where a complicating feature may disturb the typical appearance of these conditions as revealed upon the films and make it quite impossible to arrive at definite conclusions. Then we call fluoroscopy to our aid. When a shadow is being cast by a cavity, it is obvious that the excursion of the chest wall in the neighborhood of the area being studied is considerably impaired, but not altogether absent; in true pneumothorax, there being a collapsed portion of the lung, chest movements are absent, while in cases of annular pleura, there is a peculiar lagging at the involved zone which interferes somewhat with the normal expansion. When a shadow is being cast by fibroid changes, there is no appreciable change in the thoracic movement.

Autopsy evidence leads us to support Amberson's hypothesis, "that in a large majority of instances, so-called roentgenographic pleural annular shadows accompanying pulmonary tuberculosis do not represent localized pneumothoraces secondary to lung rupture, but indicate rather the presence of simple chronic or sub-

acute localized pleurisy with or without localized retraction and separation of the pleural layers". We also believe that "these shadows develop and enlarge as accompaniments of intrapulmonary tuberculosis that is usually active or progressive". Equally we endorse Dr. Honeij in his statement that pleural shadows have no relation to the extent of a pulmonary infiltration or softening and breaking down of tissue, as this is in accord with our contention that cavities are not to be expected in the absence of a well established tuberculous involvment.

Judging from our personal observation, we cannot endorse the teachings recently put forward to "consider all annular shadows as cavities until diligent investigation proves that they are to be better interpreted some other way". Why not consider them as of pleural origin in the absence of leading roentgen and physical signs of cavity? Are we to set aside the normal histology of the lung parenchyma and its logical relation to the formation of pulmonary tuberculosis excavation? Has it not been proven by serial roentgenograms that annular pleural shadows behave very differently from those of cavities? Do they not in the majority of cases disappear, leaving no trace, sometimes in a surprisingly short period of time? In one of our cases, a well developed pleural shadow which had revealed itself in serial stereograms for nearly a year disappeared in three weeks. Cavities, as we have already stated, once developed, are there to stay even though we readily admit that their size and shape may be altered during the process of repair. The acute cavity following a pulmonary abscess may heal and disappear, leaving no trace on the skiagram, but one must remember that in these cases, cavities are a consequence of inflammatory changes, and do not develop wholly at the expense of softening and true destruction of the parenchymal structure. Then, too, an acute abscess cavity once surgically drained has healthy lung tissue surrounding it ever ready to be of material aid in the process of repair. The greatest percentage of those that drain through natural channels become chronic, develop a heavy capsule and are never cured because the capsule prevents the approximation of the cavity walls, and therefore, no complete restoration is possible. Can the same be said of a tuberculous cavity? If acute, it is in contact with pathalogical tissue; if chronic, it is well walled off by a strong fibroid capsule.

The presence of fluid within an annular shadow constitutes no pathognomonic sign of pulmonary cavity; we, like Amberson and others, have drawn fluid from localized pleurisies in annular formation and have seen the pleuritic process disappear in a comparatively short time.

Artificial pneumothorax helps considerably in the diagnosis of doubtful cases of cavitation. In the presence of induced pneumothoraces, annular shadows of pleuritic origin either disappear or else, when they are of long duration and extensive adhesions have formed binding the parietal and the visceral pleuras, adhesive bands are seen strung from the visceral to the parietal layer crossing the pneumothoracic field. Cavities, on the other hand, change in position following the collapsed lung in its displacement and become distorted in appearance. When acute, they may so obliterate as to be scarcely discernible. When cavities occur close to the periphery at the mesial side of the lung, they may be so overshadowed by the median structures as not to be discernible at all. In several cases, artificial pneumothorax has revealed, by displacement and collapse of the lung tissue, cavities that had not even been suspected.

Chronic ulcerative tuberculosis may become arrested even in the unbelievably advanced cases, the gross lesion undergoing healing by fibroid proliferation and tubercle calcification. This condition is shown upon the film by the radiopaque, mottled shadows of cleancut borders already

spoken of and the irregular shadows representing the diffused fibrosis.

FIBROID PHTHISIS

Fibroid phthisis is characterized, from a pathological point of view, by an early fibroid degeneration concomitant with and following closely the infection of the organ; so that, in the early stages of the disease, the infected areas are surrounded by a capsule of fibrotic tissue, which, acting as a wall, separates the diseased portion from the neighboring healthy lung tissue. This capsule contracts and shrinks, and the infected material, sometimes caseated, within it, has a tendency to early calcification or else the tuberculous infection may be more or less generalized and consists of minute foci scattered throughout both lungs. Also, the fibroid tissue may interpose the minute foci so that both lungs may show a considerable amount of sclerotic tissue in the form of wide fibrous bands extending from the hilus through the organ to the periphery, causing occasionally true deformity of the thoracic contents. Fibroid tuberculosis is shown upon the film either by linear markings in reticular formation encircling, here and there, irregular areas of hyperdensity, early of caseative, later of calcific origin or else by heavy, dense, ribbon-like linear markings radiating from the hilus into the periphery. Ever so often, cases of fibroid phthisis are seen exhibiting all the changes described.

GLANDULAR OR HILUS TUBERCULOSIS

This is the only type of tuberculosis in the adult's chest where the aid of roentgenology has a diagnostic value, for no other disease will even simulate the shadow characteristics of the hilar structures as revealed upon the roentgenograms. We are at a loss insofar as the true patholoy of these cases is concerned, having no autopsy data to illuminate our path, but we assume that the changes are identical with those that take place in tuberculous lymphatic glands elsewhere.

For reasons that are not within our scope to explain, the constitutional symptoms in hilus tuberculosis of the adult stand out earlier and with much more intensity than in any other type of the disease; so that the clinician, in spite of lacking corroborative physical data, is more than suspicious that the patient is suffering from tuberculosis. The disease in adults has a tendency to early breaking down of the glandular structure. This does not usually cast upon the film abnormally enlarged shadows, there being but little swelling. There is an almost pathognomonic unevenness of the density of the shadow, having undefined areas of radioparency intermingled with areas of radiopacity so that the appearance is similar to the shadow cast by the breaking down of the lung tissue within a caseative zone. The sharp, well defined margins of a normal hilus are lacking, the shadow fading gradually from its center so that its borders are blurred and undefined. Traveling in all directions into the parenchyma, which has a normal appearance, short, heavy, blurred trunks are seen originating from the margins of the hilus and losing their density from within outward. They are probably being cast by infected lymph channels. In the majority of cases, in an incredibly short period of time, the disease becomes parenchymatous, physical signs make their appearance and the skiagram shows distinct mottled shadows, which spread out from the central portion towards the periphery. At this stage, a positive sputum is the rule. This is the class of cases of parenchymal infiltration in which the apices are the last portion of the organ to be involved. If the chymal involvement takes place, as it chymal involvment takes place, as it happens in a small percentage of cases, fibroid and calcific changes show themselves within the hilus, which casts a radiopaque shadow, irregular in configuration. The margins, although cleancut and well defined, have a ragged, irregular outline. The short, heavy, blurred trunks disappear, and the hilus structures stand out and form a decided con-

trast with the adjoining lung parenchyma.

We have observed these changes in the adult where the infection has invariably been more pronounced on one side. Our experience has shown that the disease behaves quite differently in children. It is equally bilateral and early swelling instead of breaking down of the structure is the rule. There is no tendency to early parenchymal involvement, and the constitutional symptoms are not as pronounced as in the adult. Then, too, in children, the roentgen signs cannot altogether be relied upon as having a tuberculous meaning, for other non-tuberculous conditions of the hilus may cast upon the skiagram similar shadows.

TUBERCULOUS PLEURISIES

We are reluctant to accept the theory that all chronic pleurisies are of tuberculous origin; scores of cases showing pleural changes upon the skiagram, accompanied by typical physical signs, have eventually disappeared, leaving no trace of either physical or roentgenological signs.

We have found that, contrary to what happens with parenchymal tuberculous infiltration, autopsy will invariably show a greater pleural involvement than roentgen shadows have demonstrated. Because pleuritic changes only cast shadows after they have attained considerable thickness. Much before this state has been reached, physical and constitutional symptoms have made their appearance so that negative x-ray findings should in no way interfere with the clinical diagnosis of this condition. We have learned that pleuritic complications are by far the rule in chronic pulmonary tuberculosis of the ulcerative type. When shadows representing pleural changes appear upon the film, they may be of great importance to the clinician. We have already discussed the leading role played by the so-called annular shadows, how they interfere with the correct diagnosis of pulmonary excavation, and their relation to the acuteness of the case. Fibrinous pleurisy intercepts radiation at such a rate as to cast an evenly hazy shadow which increases in density in proportion to the thickness of the pleural layer. Adhesive pleural changes play a decisive part in the prognostic chain. When they are so situated as to interfere with the proper excursion of the diaphragm, or so envelop the parenchymal structure as to prevent normal lung expansion, they constitute an impassable obstacle in the road to recovery. When of mesial origin, they are also a menace to the patient and are the causative factor of grave circulatory disturbances. In one of our cases, the heart and the great vessels were literally "trapped" in a heavy veil of thickened pleura so that the cardiac condition was by far the main issue.

Effusion is readily recognized when in sufficient quantity and the mediastinal organs, free from adhesions, are displaced to the opposite side. The shadow cast gradually diminishes in density from within outwards. Obviously, due to the changes in the depth of the thoracic vault, the fluid layer becomes thinner as it travels towards the periphery. The character of the fluid may increase the density of the shadow, but does not seem to interfere with the variation in density from within outwards. However, when the fluid is encapsulated, it must necessarily be accompanied by extensive pleural changes, and then the median organs are but little disturbed in their position, and the shadow is equally dense in all directions. Usually, the margins of the capsule are clearly discernible. In some cases, however, when the pleuritic changes extend beyond the capsule, the margins are lost by overshadowing. The study of fluid levels when present, either by radiography of the patient in varying positions or by fluoroscopy is of greatest value in doubtful cases. Physical signs cannot always be depended upon in the diagnosis of intrathoracic fluid. When the effusion is of considerable size, the extreme pressure of the fluid makes it act as a solid conductive agent, so

that signs corresponding to those of consolidation may be elicited.

EMPYEMA

Empyema is the exception rather than the rule in pulmonary tuberculosis. The roentgenological signs are rather conclusive, although a simple effusion accompanied by extensive fibrinous changes in the parietal pleura may cast a similar shadow. This is always denser than the heart shadow and decreases in density from without inwards. We have seen autopsies in only three cases of tuberculous empyema and in all three cases the purulent exudate was within the visceral pleura. One of these cases was so advanced that the lung tissue had totally disappeared on the affected side, having been replaced by a pus containing sac, the walls of which (the visceral pleura) had the consistency and thickness of heavy leather, with rough internal and smooth, glistening external surface. The pleural space had a normal appearance.

We have found no literature on this subject and the question comes to our mind: Are all cases of tuberculous empyema intrapulmonary? Does the infection take place, like in pulmonary abscess, and because of the low resistance and softening of the tuberculous tissues, no defensive walls are formed? The issue deserves consideration. It may influence the clinician ah to the advisability of early surgical interference.

In conclusion we will sum up a few principles referring to x-ray diagnosis in its relation to pulmonary tuberculosis. A roentgenologist should not expect his findings to be taken as conclusive, but only to be incorporated in the clinical chart as corroborative or suggestive data. In the presence of inconclusive physical signs and suspicious history, shadows representing a pulmonary lesion should be given careful consideration and should be regarded as most likely to be of tuberculous origin. On the other hand, when the history and the physical findings are not inclined towards tuberculosis, other pathological entities likely to cause similar shadows should be investigated, for one must remember that parenchymatous gummatous pulmonary syphilis casts shadows quasi identical with those seen in caseative tuberculous bronchopneumonia, that pneumosiderosis resembles upon the skiagram miliary tuberculosis, and that cardiovascular conditions may bring about pulmonary congestion, in patches and unilateral, which match closely with the early lesions of parenchymal pulmonary tuberculosis.

Our experience tells us that an etiological opinion in cases of chest radiography should be given only in the presence of unmistakable x-ray signs. Abnormal densities in the parenchyma not involving the uppers may be of tuberculous nature, but no diagnosis of tuberculosis should be made until other conditions are excluded. It is the duty of the clinician and not of the roentgenologist to determine activity. This usually requires great skill and all of the laboratory aid that can be secured. Rales over a definitely mottled or blotted out area may indicate an active tuberculous lesion, but a search into the constitutional symptomatology is essential. On the other hand, rales, even after cough, over a limited area, with negative x-ray findings, do not always and necessarily indicate tuberculosis. Is it not true that similar findings may be met with in influenza, syphilis or heart lesion? Rales heard both on inspiration and expiration, whether at the bases or at the apices, without x-ray findings, indicate possible bronchitis or early edema; prolonged high pitched expiration with increased intensity or granular breathing, both with negative x-ray findings, have little significance, but, with positive x-ray findings, they have great significance. X-ray densities described beneath the scapula or under the shoulder should not be discarded in the absence of physical findings; one must remember that these areas are dead to the stethoscope.

Speaking in behalf of and for the

art of roentgenology, we feel not at all humiliated in emphatically stating that there are limitations to the diagnosis of pulmonary tuberculosis by means of the x-ray. The words contained in this synopsis, we hope, have proven our assertion that the closest co-operation between the clinician and the roentgenologist is imperative in the proper study of pulmonary tuberculosis. In the average case, one is a complement to the other. The wide awake, progressive clinician has come to the realization that the x-ray is, without question, the best agent at his command to follow up his case, and that the roentgenologist stands out as a reliable scientific partner doing his bit towards illuminating the obscure diagnostic road.

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THE DIAGNOSIS AND TREATMENT OF PEPTIC ULCER FROM A SURGICAL STANDPOINT*

WM. H. WOOLSTON, M. D., Albuquerque, New Mexico.

The subject of peptic ulcer has been a favorite one for the pen of the gastro-enterologist. Its treatment still seems to be a point of issue between the internist and the surgeon.

The diagnosis of ulcer of the stomach or duodenum has become much more accurate since the x-ray has given us a firmer foundation upon which to work. As my preceptor, Dr. Kanavel, expressed it, "In the diagnosis of this condition, in the final analysis, we might rate the history of the patient 50 percent, the x-ray findings 47 percent, and other laboratory methods 3 percent.

A carefully taken history is more important in the direct diagnosis of peptic ulcer than any other agent we have.

Localized epigastric pain, the one most important symptom, is characterized by its chronicity, intermittency and periodicity.

It is chronic in that it exists for months to years. Peptic ulcer is almost always chronic by the time it causes symptoms. Robertson of Rochester and numerous other pathologists have found healed gastric and duodenal ulcers during the course of autopsies on patients who had died of other causes and whose histories did not reveal stomach symptoms.

It is intermittent in that the symptoms last from a few weeks to six months and then disappear with or without medical treatment. This is followed by a period free from pain lasting from a few months to two years. The more advanced, or the more chronic the lesion becomes the shorter is this "free" interval.

It is periodical because the pain and distress appear with regularity, following meals from one-half to two hours, depending on the location of the ulcer.

These three characteristics are most constant. Other attributes of the pain are food and alkali relief. Epigastric pressure relieves it temporarily. It is a gnawing, burning pain, felt most frequently when the stomach is empty.

Other signs are localized tenderness, hematemesis, melena.

RADIOLOGY

Without the x-ray, unless one has a well established clean cut ulcer, the diagnosis as it depends on the symptomatology is quite likely to be inaccurate. As Sir Berkeley Moynihan, in reference to peptic ulcer, has written, "The radiographic method is, in expert hands, the one certain method of diagnosis and is nod an indispensable

(*Read at the Ninth Annual Session of the Medical and Surgical Association of the Southwest, at El Paso, Texas, Dec. 11-13, 1923.)

addition to the older and less accurate methods of inquiry."

By fluoroscopy we note the position and outline of the stomach and determine whether there are filling defects, spasms or sulci, and their characteristics; whether there is fixity or deformity of the bulb.

The most accurate roentgenographic method is by serial plates with the observation of the morphology of the stomach and persistent defects, such as niches, accessory pockets, incisura, craters, hour glass deformities, dimpling, diffuse spasm, and the residue after six hours.

The radiographic characteristics, as well as the various radiologic types of ulcer, we shall leave to the discussion of the roentgenologist. They include, for instance, penetrating, burrowing, large shallow, small round, and healed ulcers.

In addition to the above mentioned factors in the diagnosis, there is the fractional test meal, Dr. Einhorn's string test, blood in the stomach contents or stool.

In the differential diagnosis we may ask ourselves first whether the ulcer is gastric or duodenal, and here we have the time of appearance of the pain and the x-ray to help us out. Among other diseases of the stomach carcinoma is the most important. The mode of onset, the age of the patient, character of the pain, loss of weight, vomiting; late in its course the tumor mass, exploratory operation, and last but not least the x-ray, are the cardinal points in establishing this condition. An ulcer projects as an addition to the stomach while cancer encroaches upon the lumen of the stomach. Another condition within the stomach which may be confusing, though it is not common, is syphilis.

Among diseases of adjacent viscera leading to disturbances of gastric function is gallbladder disease, which is differentiated by the character and course of its pain, radiologic findings, jaundice, chronic indigestion and colic. Spasmodic or chronic appendicitis causes stomach disturbances not unlike gallbladder disease or ulcer. Here again a carefully taken history and our friend, the radiologist, lead us to the cause.

Cirrhosis of the liver, chronic alcoholism, pernicious anemia, gastric disturbances as associated with tuberculosis, pancreatitis and other causes of abdominal pain too numerous to mention may be ruled out by careful investigation. Remember the attributes of ulcer pain and let the radiologist do his work!

TREATMENT

When we come to the treatment of peptic ulcer we approach the mooted point between the internist and the surgeon. After hearing a symposium on peptic ulcer at the Albuquerque meeting last summer we were almost convinced that the role of the surgeon in the treatment of ulcer had become a minor one. One surgeon of some fame in the west passed the palm to the internist. In slang parlance he had "tossed up the sponge". However, many surgeons have not reached that point. I believe that these cases should be treated in co-operation with the internist, for a regulated diet and medical therapy postoperative is of great importance. At least until the altered physiology has become adapted to new conditions and surgical wounds in the viscus have become entirely healed.

Medical treatment as advocated by Sippy, Einhorn and others is applicable to the acute or small round mucosal type of ulcer, especially in the duodenum. Since we are still in the dark regarding the etiology, medical treatment must still be more or less empirical. The theory of Rosenau that it is due to a strain of streptococci emphasizes the relation to it of focal infections. When I was on the pathological service at the Cook County Hospital, we made blood cultures on about twenty-five cases of peptic ulcer and in seventeen found a streptococcus. We injected a suspension of these into the blood stream of rabbits, but were not very successful in reproducing ulcers, though in two cases we found a distinct ulcer of the stomach, and in two others sub-

mucosal hemorrhages. Carlson, of the University of Chicago, in his study of the physiology of the gastro-intestinal tract, can relieve ulcer pain by increasing the gastric acidity or by fluid or food. It is a well known fact that there are remissions in ulcer pain lasting from months to two years, and, as stated above, pathologists have found evidence of ulcers which have healed without treatment of any kind. Therefore, we have no proof of a specific cure for ulcer by medical means. We must grant, however, that there is a great deal of value in the alkali and dietary methods, which give the stomach rest and allow the lesion to heal, some permanently and others temporarily. As the old fellow said, "Let Nature do its perfect (?) work". An ulcer healed in this manner leaves a scar. The mucosal borders do not approximate in the healing process. Since the etiologic factor remains, and the site of the previous ulcer remains as a local focus of lowered resistance, the recurrence of the ulcer at the original site is not unlikely when medical treatment is discontinued. We might cite the varicose ulcer of the leg as an analogy. As long as the varicose veins remain, with scars of previous ulcers, there are frequent recurrences after repeated healing.

One of the first surgical procedures was the gastro-enterostomy, which has fallen somewhat into ill repute, largely due to its indiscriminate use, especially when an ulcer cannot be demonstrated, which may result in a disordered digestion which did not exist before. There is no set surgical procedure which can be followed in all cases. Posterior gastro-enterostomy with ligation of the pylorus in duodenal ulcer with or without resection of the ulcer, ruptured ulcer with suture of the leak, pyloric obstruction or cases in which adhesions are so dense as to obviate other measures, are its chief indications. Dr. Hans Finsterer of Vienna has practically discarded the gastro-enterostomy in favor of gastric resection except in cases of perforation or acute hemorrhage many of which he states will stand resection.

Large callous or indurated ulcers at or near the pylorus may be treated by a Billroth I, the Polya, or Polya-Balfour, with resection of the pylorus. The Polya operation with anastomoses of part of the resected end of the stomach to the side of the duodenum was a method we usually followed in this type of ulcer in Dr. Kanavel's clinic at Wesley Hospital, Chicago. The free end of the duodenum was attached to the side of the lesser curvature with a few interrupted sutures. We felt that this method was less likely to leak or cause trouble than the Billroth I, though many do the Billroth I without difficulty. The Finney pyloroplasty, or Horsley's operation, have their indications, the latter being a very successful method of dealing with these conditions. Judd's method of resecting indurated gastric ulcers is well known. A resection of the ulcer with the surrounding indurated tissue should be done whether or not we believe a peptic ulcer undergoes carcinomatous degeneration, since a callous ulcer is often indistinguishable from an early carcinomatous ulcer. A sleeve resection in hour glass stomach seems to cause less difficulty and alter the physiology less than the V-shaped resection and, as Judd says, the gastric acidity is much diminished. Dr. Finsterer's mortality in 407 cases has been 4.4 percent and in the last three years 1.8 percent in 158 cases in which resections were done. His work is largely done under local anesthesia with infiltration of the celiac plexus. The death rate with conservative methods including perforations and hemorrhages is probably not less than this. In the first twenty-four hours after a perforation surgical results are fairly good. With a longer period the death rate is high. Dr. Karl Meyer of Chicago has recently written a paper on the diagnosis of impending perforation and operation which is getting the diagnosis down rather fine. An infected gallbladder or appendix should

be removed as a possible focus of infection.

A medical and dietary regimen following operation until the stomach has accustomed itself to the altered physiology is vitally necessary to the successful surgical treatment of ulcer. As stated before, we grant that the acute or subacute peptic ulcer with little or no induration falls within the internist's domain, but to procrastinate with the chronic indurated or callous ulcer, in which such serious complications as perforations, hemorrhage and malignant degeneration are lying in wait, is to court disaster.

DISCUSSION

DR. C. A. THOMAS, Tucson: Would like to express appreciation of the paper and try to emphasize a few points. I consider etiology the most important point in a discussion of peptic ulcer, whether regarded as a surgical or medical condition. It should be regarded as a secondary disease. Many experiments have been performed in the effort to produce peptic ulcer, and we have found that probably no one factor is entirely responsible; traumatism, even to the blocking or destruction of four-fifths of the circulation to an area, does not regularly cause ulcer; we must have other factors in addition to the traumatism, and among these factors we must have infection. That is why I say that ulcer is a secondary disease and we should never leave any factor uninvestigated; for this reason, the elimination of foci of infection (teeth, tonsils, appendix, etc.) is necessary, and before surgical treatment is instituted, all these things must be dealt with. If organic deformity or obstruction is present, that may demand surgery. In surgery of the stomach, we should not be satisfied with a simple gastro-enterostomy, but the ulcer should be dealt with. A simple gastro-enterostomy would result in a cure in only 25 percent of the cases; other procedures would be required in the larger majority.

DR. J. C. MASSON, Rochester: The author has taken up the important points and will mention only one or two others. As the last speaker said, the part that infection plays in peptic ulcer is a real one. Am a great believer in Rosenow's ideas of the selective affinity of bacteria, and if anyone sees his experiments they will believe. One such is to take the pus from an acute appendix and inject it into an animal or into a large series of animals and then find definite evidence of acute trouble in the appendix, and many of them with trouble in the stomach, gallbladder or duodenum, with the same type of bacteria affecting more than one organ in the gastro-intestinal tract. In all cases of peptic ulcer, the appendix should be removed, if it shows any signs of trouble, because it probably harbors the same infection.

Under the diagnosis of peptic ulcer, the author gave credit to the x-ray as being a real help in 25 percent of the cases. We believe that this should be much higher. There is no doubt that the clinical history will usually indicate ulcer, but the x-ray man doing a great deal of gastro-intestinal work will have a very small percentage of error. At the clinic the x-ray is positive in about 95 percent of the cases; that is, they miss only about five percent of the ulcers which are afterwards proven to be present.

With regard to the treatment; the last doctor, in discussion, said that probably 25 percent of the cases would be benefited by gastro-enterostomy; our experience with duodenal ulcer is better than this; eighty percent of them, without anything else except the gastro-enterostomy being done, have been entirely satisfactory, as shown by our own follow-up system and that of the New York Life Insurance Company. We are doing more treatment to the ulcer locally at the present time. Statistics show that about 85 percent of these cases are satisfactory and that gastro-enterostomy alone compares very favorably with gastro-enterostomy and excision. We have gotten away from the use of permanent suture material, which is the principal cause of gastro-jejunal ulcer.

In gastric ulcer, there is no question about the excision, because we think there is a predisposition to malignancy, and we know that gastric ulcers become malignant in a much larger percentage than duodenal ulcer; therefore, the local ulcer should be excised.

DR. CARHART, Fort Bayard: Wish to make one point by way of warning to the internist. The majority of men believe in the Sippy method of alkalinizing the stomach contents. There is a condition resulting from the excessive use of alkalies which is just as grave as any of the symptoms due to ulcer or to the acidity. That is the condition of alkalosis. The heavy use of alkali is not without danger. A patient who has a low H-iron condition of the tissues and fluids is the type of case which will develop alkalosis. The complete alkalinizing of the stomach contents is wrong and not based on physiological grounds. All that is necessary to put the ulcer at rest is to reduce the normal physiological function to a minimum without neutralizing entirely with alkali. It will repay you to look up the literature on alkalosis.

DR. W. O. SWEEK, Phoenix: The question as to when a case becomes a surgical case should be left to the internist to answer. That responsibility should be the internist's. A gastric ulcer is not the surgeon's business until the internist says so.

DR. R. D. KENNEDY, Globe: We see so many of these cases who have had attacks extending over a number of years. Perhaps the internist can cure many ulcers, but what hope has the internist in curing the cases where induration exists?

DR. WOOLSTON(closing): In replying to Dr. Masson; we figure the value of the various diagnostic approaches on the basis of 100 percent, giving history 50 percent, x-ray 47 percent, stomach analysis, etc., 3 percent. With regard to the Sippy treatment; if you accept Carlson's latest ideas, it knocks the Sippy method in the head; he says there is no disease known capable of producing a true hyperacidity. It must be further remembered that in gastro-duodenal ulcer, there may be normal acidity, so-called hyperacidity or complete achlorhydria. In 27 cases of proven gastric ulcer there were four with achlorhydria and four with low HCl. If the medical treatment depends for its cure on neutralizing the stomach contents as continually as possible, it cannot relieve if the hyperacidity does not exist.

CASE REPORT

DR. W. L. BROWN, El Paso, Texas.)

An unusual case of spider bite: Patient male, age 40, weight 195 pounds, section foreman, and lives at a station on the desert.

On the evening of December 10 he took some pills, and about five in the morning they acted on his bowels. He went to a surface toilet, which had not been much in use and was full of cobwebs beneath the seat. While there he was stung on the end of the penis.

His description of what occurred is as follows:

No puncture or local swelling was visible at any time. The sting felt like a touch ot acid, with very little pain at that time. In about fifteen minutes severe pain developed within six or eight inch radius. From the waist down a nervousness was felt, which rapidly developed into severe pain and jerking of the limbs.

Pain began to move upward, but never seemed to reach higher than the chest. At the beginning of the severe pain profuse perspiration began and continued for about seventy-two hours, with a strong vinegar-like odor; also a scent of ammonia was noticeable when bed covers were raised.

He arrived at the hospital at 7:50, about two hours and fifteen minutes after the accident. At that time the pain was so agonizing that it was impossible for him to be still a sufficient length of time to get a hypodermic. He was still perspiring profusely. He was given one-quarter of a grain of morphine, and in twenty minutes this was repeated. At this time, while his hands and feet were very cold, he complained of severe burning in soles of his feet. Also at this time he had severe pains in his abdomen; in fact, all over. He was also extremely nervous and showed evidence of fear. At 4:30 p. m. it was necessary to have another quarter grain of morphine, and another at 5 a. m. the following morning, making one grain of morphine during the twenty-four hours. He required no morphine after the first twenty-four hours, but continued in profuse perspiration. Some of the attendants thought the perspiration had the odor of acetic acid. He only vomited once, at one o'clock, on the day he entered the hospital. By the evening of the first day his abdomen was decidedly distended, and considerable difficulty was experienced in moving his bowels during the following twenty-four hours. On the second morning following his entrance into the hospital he was put on full doses of bromides for the nerves. That evening he developed a diffuse dermatitis, which caused a great deal of burning and looked like a severe case of scarlet fever, except that the rash did not involve his neck and face. He left the hospital on the seventeenth, five days after entering, looking hollow-eyed, like he had lost ten or fifteen pounds of weight, with still some burning in the soles of his feet, and quite nervous. There was never any evidence whatever of a sting locally at any time he was in the hospital; was never any local swelling, nor discoloration. The only localizing indica-

tion of the sting was the local pain.

Immediately after the spider bite the patient looked into the toilet and saw a small black spider in the web. This spider was observed there each day for six or seven days. No other spiders were observed. It was caught alive, and was found to be the Latrodectus mactans, known as the "black widow," "shoe button spider," and Po-ko-moo spider.

The following is the description of the spider, from "Poisonous Animals of the Desert," by Charles T. Vorhies:

There is a certain species of small, black spider, about half an inch long—the "black widow," or "shoe button spider," Latrodectus mactans, which is seemingly a somewhat formidable exception to the general rule concerning small spiders. This kind is reported from "the South," not specifically Arizona, but doubtless exists here. Experimental evidence shows that this other species of Latrodectus from various parts of the world have a much more virulent poison than ordinary spiders. This is borne out by reported cases, while in the various countries where they are native these spiders are almost universally held to be dangerous.

The Latrodectus mactans (called Po-ko-moo by certain California Indians) is, according to Merriam, as quoted by Comstock, ranked by them "with the rattlesnake as poison. To poison their arrows they mash the spider and rub the points of the arrows in it." The symptoms produced experimentally agree with the symptoms of persons who have been snake bitten. There are several cases on record of severe symptoms following the bite of this species, one of which ended fatally. This may even then have been an exceptional case due to personal condition or even in part to fear. As I read over the account of this and another severe case from the same locality (North Carolina) I cannot but wonder whether the reported dosage with alcohol may not have put the fatal touch upon the one case and the nearly fatal touch upon the other. We know now that whiskey is the worst thing to give at certain stages in rattlesnake bite, notwithstanding popular opinion to the contrary, and it may well be so in the case of the bite of this spider. A case is personally reported to me from Yuma, Arizona, of a bite from a black spider, doubtless this or a related species. In this case the victim, a lady, was bitten on the hand and serious "symptoms of blood poisoning" followed, which it required some weeks to cure.

This exceptional spider is easily recognizable, being shiny black, with red or yellow markings. An hour-glass shaped mark on the lower side of the abdomen is the most constant of these marks. Spots may in addition be found along the middle of the back. The male has in addition to these, four pairs of stripes on the sides of the abdomen. A gray species of this genus has been described from California, and may belong to our fauna, but no reports are available concerning injury from it.

Discussion by DR. PAUL GALLAGHER: There is no positive evidence that the cause in this case was a spider, but from one case which I saw, where a spider was the undoubted cause, I believe that it was the same here.

Several years ago a man presented himself with a small spider which he had killed. It was impossible to determine its characteristics, as it had been mashed, but it was evidently a black spider about the size of the small fingernail. During defecation he was stung on the penis. No local lesion of any sort developed at all. But beginning almost at once he had severe pain in the groin and testicles. He said this pain felt as though one were invaginating the testicle through the scrotum into the inguinal canal with a hot iron. As the pain in this area subsided somewhat he developed pain across the chest, constricting in character, but not interfering with respiration, and a severe pain in the back. He then developed pain in the legs and burning in the soles of the feet and in the palms of the hands. The pain at all times, until suppressed by morphine, was excruciating in character.

In describing this pain later, he stated that at one time he had been in an automobile accident where the machine fell upon and fractured one thigh, pinning him to the ground. When help arrived, the car was jacked up and twice the pack slipped, letting the car fall back upon the thigh. He believed that this pain was much more severe than that which he felt when the car fell from the jack upon the broken thigh.

He required in all six quarter grain tablets of morphine at half hour or hour intervals to relieve his pain; all were given between eight in the morning and five in the evening. After relieving his pain, he recovered very promptly so that at the end of twenty-four hours he was well again except for terrific soreness of all his trunk muscles.

X-RAY MACHINE FOR SALE

A Victor bedside x-ray unit, thirty millampere, with 30 m.a., radiator Coolidge tube, in excellent condition. Can be purchased at a very attractive figure. Excellent outfit for a small hospital or doctor who is not located where there is an x-ray laboratory. For further particulars, write Box 1328, Phoenix, Ariz.

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DR. H. S. BENNETT
Fort Bayard, N. M.

Dr. H. S. Bennett, of the United States Veterans' Bureau Hospital, at Fort Bayard, N. M., died on February 3rd, following an operation for obstruction of the bowels.

Dr. Bennett was born in New York City and was 47 years of age at the time of his death. He took his bachelor's degree at Princeton University and his medical degree at the College of Physicians and Surgeons in New York City, practicing in Washington, D. C., for a number of years. He was connected with the U. S. Public Health Reserve during the epidemic of 1918, and had been connected with the Veterans' Bureau, as a specialist in heart and chest diseases, for the past three years, being located at various times at Oteen, N. C.; Alexandria, La., and for the past year had been at Fort Bayard.

Dr. Bennett, on arriving at Fort Bayard, immediately showed an interest in organized medical affairs, which was very gratifying to the profession of New Mexico. He helped revive and reorganize the Grant County Medical Society and was secretary of the organization at the time of his death. He was a guest of the Medical and Surgical Association of the Southwest at their El Paso meeting in December, reading a very scholarly paper on "The Heart in Tuberculosis", which was printed in the February issue of this journal. He was enlisted in the plans for enlarging the scope and influence of the Southwest organization, and his death is a distinct loss to the medical profession of the entire southwest, where men who are genuinely interested in organized professional efficiency are all too few.

Burial was made at the cemetery at Silver City, N. M.

DR. HOWARD THOMPSON (El Paso) RETIRES

Dr. Howard Thompson, the grand old man of the El Paso County Medical Society, in the 74th year of his life, rests on his oars in retirement from the practice of medicine.

Dr. Thompson graduated from the Medical College of Ohio, in Cincinnati, in March, 1881, practiced from that time until April, 1885, in Franklin, Ind., then came west and was located at the Mescalero Indian Agency for four years. Following a term of postgraduate work at the University of Cincinnati, he came to El Paso in March, 1890, and for 25 years had the same office in the building the site of which is now occupied by the State National Bank.

Dr. Thompson was the fifth president of the El Paso County Medical Society, serving from 1901 to 1902.

For over thirty-three years he served well this community as a very active and capable physician.

Since his retirement, Dr. Thompson has returned to the Mescalero Indian Agency, where he is enjoying his more than three score and ten years in good health and happiness.

WANTED—A Physician to locate at Reserve, N. M., the County seat of Catron Co. For information write to Hugh Fraser, Reserve, N. M.

EL PASO COUNTY MEDICAL SOCIETY REPORTS

Regular meeting of the El Paso County Medical Society was held at the University Club December 3, 1923.

The meeting was called to order at 8:10 p. m. by the president and minutes of the previous meeting were read and approved. There were 36 members and 3 visitors present. The president in an announcement drew attention to the closeness of the meetings of the 10th and 17th to that of the Medical and Surgical Association of the Southwest, December 11-12-13, with the probable selection of December 17th as the annual banquet date of this society, the closeness of the 24th with Christmas, and the 31st with New Year's, asking what the society desired to do with regard to having meetings on those dates. Motion was made by Dr. W. L. Brown and seconded by Dr. Paul Gallagher to have no meetings until after the first of the year. Motion was carried.

There were no case reports. Dr. H. H. Stark read a paper, "The Field of Vision", which was illustrated with 24 slides. Discussions were made by Drs. J. M. Britton, J. W. Davis, F. P. Schuster, E. A. Duncan, Hugh Crouse and E. H. Irvin.

Dr. R. B. Homan made an announcement, explaining the efforts of the State Medical Association to create a special fund of $5 per member which is to be devoted to educational purposes, and the selection of prominent men throughout the state for literature. He asked the president to create a committee to raise the quota for this society. Dr. Waite made motion that such a committee be created and that Dr. R. B. Homan be made its chairman; seconded by Dr. J. W. Laws, and carried.

Dr. J. W. Laws offered two resolutions, one in behalf of Dr. John W. Tappan and the other Sanitary Engineer R. E. Tarbett, United States Public Health Service, expressing the appreciation of this society for their invaluable efforts in the mosquito campaign. The motion was carried.

Dr. W. L. Brown made motion that resolution be drawn of a similar character for Dr. Hugh Crouse; seconded by Dr. J. W. Laws, and carried. In a statement that what he had done was for the society and his city, Dr. Crouse modestly asked that such action not be taken. He was, however, overruled. Dr. W. L. Brown and Dr. J. W. Laws were appointed members of the committee to draw up such resolution.

The president called for committee reports as to the advancement of arrangements for the meeting of the Medical and Surgical Association of the Southwest. It was noted that everything is in readiness.

Dr. Hugh Crouse outlined six talks that he has been requested to give to the teachers of the El Paso schools (numbering about 900) on public health, prevention of disease, etc., and asked suggestions from the members. It was conceded that he had covered the field completely, and no suggestions were offered.

Next in order of business was election of officers, which was done by nominating by informal ballot and election by ballot. Those nominated for president were Drs. J. A. Hardy, W. W. Waite, C. P. Brown and E. B. Rogers. Second ballot was drawn, in which Dr. Waite received 19 votes and Dr. Hardy 17. This not being a majority of the members present, Dr. Waite was elected by acclamation.

For vice-president there was but one nomination made, and that was for Dr. J. A. Hardy, who was elected by acclamation.

For secretary-treasurer the nominees were Drs. F. P. Schuster, W. R. Jamieson, F. O. Barrett and M. I. Leff. On ballot Dr. Barrett received 16 votes, Dr. F. P. Schuster 9, and Dr. W. R. Jamieson 1. Dr. Jamieson made motion that Dr. Barrett be elected by acclamation, which was done.

For librarian Dr. E. A. Duncan was nominated and elected by acclamation.

For delegate to the Chamber of Commerce, Dr. J. W. Laws was nominated by ballot and elected by acclamation.

Censors: It was an unusual feature that, due to the resignation of Dr. B. W. Wright and the death of Dr. W. R. Smith, who were the second and third year appointees, all three of these positions became open. Nominations for these three offices were made on the same ballot by ruling of the president, the doctor receiving the highest vote to serve three years, next highest two years, and third highest one year, and the following were elected by acclamation: Dr. S. T. Swope for three years, Dr. Harry Leigh for two years, and Dr. Paul Gallagher for one year.

For milk committee, Drs. Craig and Safford were the successful nominees and were elected by acclamation.

For representative of the American Society for the Advancement of Science, Dr. E. W. Rheinheimer was nominated by unanimous vote and elected by acclamation.

No other business being brought before the house, motion to adjourn was entertained at 10:40 p. m.

Minutes of January 7th meeting were published last month.

A regular meeting of the El Paso County Medical Society was held January 14, 1924, at the University Club.

The meeting was called to order by Dr. W. W. Waite, president, at 8 p. m. There were present 24 members and two visitors.

Owing to the absence of the secretary-treasurer, the reading of the minutes of the last meeting was dispensed with.

Dr. Crouse presented a very interesting case of spastic gait, a man about 28 years of age presenting the appearance of one aged 40. Discussions were made by Dr. Swope and Dr. S. A. Schuster.

There being no further case reports, the program, in charge of Dr. Harry Leigh, fol-

lowed. The symposium on "Congenital Syphilis" was opened by Dr. Leigh, whose paper was illustrated by slides showing different facial phases of the disease in children.

Dr. Henri Letord, dental surgeon, told how the teeth are affected in cases of hereditary syphilis, showing slides of Hutchinson teeth and others.

Dr. F. P. Schuster discussed the subject from the standpoint of the eye, ear, nose and throat.

Dr. P. E. McChesney spoke of the neurological results from congenital syphilis and told of the large number of cases found in the examination of school children.

The symposium attracted a great deal of interest and was liberally discussed by Dr. S. A. Schuster, who brought out the fact that in nearly all cases which had come to his attention at the clinic, there was a secondary infection and that anti-syphilitic treatment alone was not sufficient in such cases.

Major Scott, in discussion, told of the value of the metallic bib, in treatment of congenital syphilis, when another cloth, saturated with mercurial ointment, was slipped in the bib.

Dr. Waite brought up a number of points which had not been mentioned by Dr. Leigh in his paper, and asked just how far hereditary syphilis could be transmitted—if transmitted to the third generation, would that person transmit it to his or her offspring?

In closing, Dr. Leigh stated that there were a great many points upon which he had not attempted to touch in his paper, and told of a case he had had several months ago, which was undoubtedly congenital syphilis in the third generation.

The board of censors, Drs. Swope, Leigh and Gallagher, submitted approval of the application for membership of Dr. J. E. Wright, Alpine, Brewster County, Texas. It was moved and seconded that Dr. Wright be accepted for membership and the motion unanimously carried.

No further business coming before the chair, the meeting was adjourned at 9:55 p. m.

Regular meeting of the El Paso County Medical Association was held at the University Club, El Paso, Texas, January 21, 1924.

The meeting was called to order by the president, Dr. Willis W. Waite, at 8:10 p. m. There were present 23 members and 3 visitors.

The minutes of the last meeting were not read, the president announcing that this would be dispensed with if it met with the approval of the members.

There was one announcement—the invitation of Messrs. Peak and Hagedon to the official opening of their new establishment.

No clinical cases being presented, Dr. Crouse was asked to proceed with his symposium on the "History of Medicine". Dr. Crouse gave a very brief outline of medical history, which was followed by a paper by Dr. E. A. Duncan, entitled "The History of Percussion and the Life of Auenbrugger", who inaugurated the method of diagnosis. Dr. Crouse then gave a very interesting impromptu talk on the history of auscultation and Laennec, who devised this method of diagnosis and invented the stethoscope.

Discussion was opened by Dr. S. D. Swope, who, in the course of his talk, mentioned the discovery of a skull in one of our western states, which had a trephine. This was the first of its kind to be found in North America, many having been found in Central and South America. The interest attached to this incident is the evidence of very early usage of this type of surgery.

Further discussion was entered into by Drs. Leff, Egbert, Laws, Capt. St. Peter, Drs. Riley, Werley, Gallagher, Leigh, Darnell and Waite, and discussion was closed by Dr. Crouse.

Report of the committee to investigate the advisability of a permanent home for this society was made by Dr. Gallagher, who stated that in the First National Bank Building three rooms, which could be turned into one, could be procured for $70; that the cost of the furnishings would be about $200, and that the Dental Society would agree to hold its meetings there at $10 per month, which would make the rental $60 per month. He did not feel that the society should assume the additional indebtedness oer what it is now paying, which would be about $15 per month.

Dr. Werley made motion, seconded by Dr. Hardy, that the committee be continued and make additional report. This was carried and Dr. Gallagher resigned from the committee, stating that he was not in favor of making a change.

The president announced the appointment of the legislative committee, as follows: Dr. Strong, Dr. Rheinheimer and Dr. E. B. Thompson.

The president also announced the holding of the annual meeting of the Southwestern Medical and Surgical Association of the Southwest during the coming fall in Phoenix and urged that as many papers as possible be written by members of this society and that oral requests to be on the program be made in order that this society might be well represented and help in the success of the meeting.

He announced further that papers for the meeting of the State Association to be held in May must first be read before the County Society and reported to the state organization not later than March 25, 1924.

As there was no other business, motion to adjourn was entertained at 9:57 p. m.

Regular meeting of the El Paso County Medical Association was held at the University Club on January 28th.

The meeting was called to order by the president, Dr. Willis W. Waite, at 8:10 p. m., there being 19 members and 1 visitor present. Reading of the minutes of the last

meeting were dispensed with. Clinical cases were then called for in accordance with the program prepared for the evening.

Case Report by Dr. Meyer I. Leff.

The following interesting case of extrapleural thoracoplasty, the first case of its kind to be performed in the United States, was reported by Dr. Meyer I. Leff:

L. S., male, age 30, at time of operation had been suffering from pulmonary tuberculosis six years previous to operation. Climatic and hygienic treatment did not relieve it. He was running a temperature between 103 and 105, heart very rapid and had frequent hemorrhages. Left lung was full of small cavities, sputum profuse and laden with tubercle bacilli. Patient was losing ground rapidly.

First operation, extrapleural thoracoplasty, for permanent collapsing of the left lung, was performed by Dr. Willie Meyer, New York, in November, 1918, at which time portions of the tenth to fourth ribs, inclusive, were removed. Second operation was performed December 5, 1918, three weeks after the first. Portions of the fifth to second ribs, inclusive, were removed.

Both operations were performed under local anesthesia. After the first operation, patient's temperature came down; after the second operation, the cough was improved, sputum diminished in quantity and free from tubercle bacilli. The size of the removed portions varied between 7 inches from the tenth rib and 2 inches from the second rib. Patient has made a complete recovery, has been working for the last three years and is clinically cured.

Case Report by Drs. Hugh Crouse and G. Werley.

DR. CROUSE: I am going to report a case, dividing it into two parts, allowing the doctors present at the conclusion of the first part or division to make a guess as to the diagnosis, basing their opinion on the history as given in the primary division of the report. The second division of the case history will be handled by Dr. Werley, and at its conclusion he will give you the diagnosis.

A man, 49 years of age, had not had a sick day in 20 years, does not drink, smoke or use tobacco in any form, keeps absolutely regular hours. Friday at noon had a lunch consisting of soup and cold slaw, was taken with intense pain in the sternal region, upper third, shortly after eating. I saw him within 30 minutes from the time the attack began; pulse 72, perfectly regular, yet the man was absolutely fighting for air and had the facial expression of intense pain. Temperature absolutely normal. This man, a Spanish War veteran, with lots of nerve, was not shamming. I assumed it was an acute aortic dilatation, with its consequent sympathetic nerve irritation and reflex to the right arm. I gave him ¼ grain morphine, shortly afterwards 1/30 grain apomorphin, after which he vomited about a gallon. This vomitus was very red, but his wife said he had been eating tomatoes. His vomiting did not seem to relieve his pain. I gave him another dose of morphine, with no relief. Finally I had to administer chloroform in order to afford him relief. If you touched his chest to the right of the sternum about the fourth rib, it was so intensely painful to him that he would cry, "For God's sake, stop!" About four o'clock he vomited again. This was a liter of seemingly diluted blood. Two hours later he vomited again, seemingly pure blood. His vomiting was repeated at varied intervals in gradually decreasing quantities until four o'clock the subsequent morning. In the evening his pulse became slightly irregular, reaching the maximum height, 110. No sounds over the chest indicating any pulmonary involvement except for a slightly increased bronchial breathing near the hilus of the lung. He was treated with morphine and absolute quiet, not letting him up and giving him nothing but cracked ice by mouth.

What was the matter with him?

A further point; two weeks before this attack, his blood pressure was 130 and he passed life insurance examination for $10,000.

Dr. Waite suggested pulmonary thrombosis. Members present asked if x-ray had been made and were informed that this had been impossible, as yet. Numerous conjectures were made as to the diagnosis in the case by various members present.

DR. WERLEY: I first saw the patient Saturday night. Dr. Waite made a leukocyte count and found white count 20,000 polys 87; red count 4,400,000 hemoglobin 75 percent. Temperature had gone up a little, running from 99 to 100.8, fever continuous. He had a great deal of dyspnea, with respiration that was somewhat irregular and just a little rapid, about 24, sometimes as high as 26, sometimes 20, and while he did not have to sit up to get his breath, he was not satisfied with what he could get and said he could not fill the upper part of his chest. About the time I saw him he was comparatively easy, but it was only about Sunday night that we could leave off morphine. He has told me since that he called for morphine before he had pain, for fear the terrific pain would come back again, and therefore he wanted to have the morphine in plenty of time. On going over his chest, I found heart sounds quite readily heard at the base of the heart, with the second pulmonary sound a good deal accentuated. The heart sounds were heard without any great difficulty, but they were certainly somewhat feeble. A diffuse impulse could be felt slightly over the apical region of the heart, but it was impossible to locate the apex at any definite point. The outline of the heart was enlarged. The arch was 6 cm. wide. The right side was 4½ cm. to the right; apex wall beyond the nipple line at least almost 2 cm. The liver could not be felt; spleen could not be felt; abdomen was somewhat distended with gas, but was soft. At one time he complained of a little pain over the appendix. I pressed down

deep and there certainly was not anything there. He said he thought maybe he had strained himself. I listened to the lungs, but there was nothing to be heard abnormal except at the right base below the scapula over a wide area there were very definite crackling rales at the end of inspiration. He still has these rales, but not as marked as they were. At the present time heart apex has gone back inside the nipple line. Sunday at 6 a. m., Dr. Crouse was called to see him and called me. At that time he had a squeaky loud to and fro sound synchronous with the heart beat, disappearing as it went away from the heart, but which could be heard over a wide area. When I went back at night, this had disappeared. Blood pressuer was 114 S., 74 D. When he was well his pressure ran about 130, so it was quite evident there was a definite fall in blood pressure.

My diagnosis, cardiac infarct, is based on what I have read and seen about such cases. The points in the diagnosis of cardiac infarct are: First, you have pain, a type of angina pectoris, but in cardiac infarct there are certain points more or less diagnostic—a little fever, a drop in blood pressure and dyspnca is quite characteristic, whereas, in angina there is a rise in blood pressure and no dyspnca. A third point in the differentiation is that the pain persists for at least 48 hours and is severe, whereas, in the ordinary case of angina pectoris it is often very brief and only for a few seconds or minutes, at least not over a half hour, as a rule. It usually passes quickly, while the infarct pain is something you cannot get rid of. You give ¼ grain of morphine and it does not relieve it. It was necessary to give this patient chloroform before he finally got at all comfortable. The pericardial friction rub is of great diagnostic value. Also there is an increased leucocyte count which is not found in an ordinary angina.

Hamburger reports two cases of angina pectoris of the abdominal type, associated with jaundice. He says a number of cases reported have had gastric hemorrhages with the attack. So the fact of a gastric hemorrhage does not prevent us from making a diagnosis of cardiac infarct. I have seen such cases myself. There is much danger of cardiac infarct being mistaken for some surgical condition of the abdomen.

I think when we come to watch for these things we will find they are very much more common than we think. There is nothing that I know of except infarct of the heart that causes anyone to die without a struggle. That differentiates it from apoplexy. Another diagnostic point in these cases is that nearly always you will find rales in the chest. That is due to the left heart being disabled.

Case Report by Dr. E. J. Cummins.

I have a case to report tonight. I believe it is the second case of its nature to be reported before the El Paso County Medical Society, the fourth case of its kind to be reported in Texas, and the eighth case to be reported in the United States and Canada. It is a case of mycetoma or Madura foot.

This patient, a Mexican woman, 28 years of age, always lived in Mexico until quite recently and is the mother of three children; two died in early childhood—we do not know from what disease; the last child a baby three weeks old, living and well. Nine years ago she says she felt something in the sole of her foot as if there was a sliver in it and she picked it with a needle, trying to get the sliver out, but was unable to find anything. Shortly after that a nodule appeared there, which gradually grew, and then other nodules appeared. Up until two months ago she went around walking perfectly comfortable, but since that time the growth was very rapid until it reached its present size. I have a specimen here and some pictures of the woman. The first one is a photograph of the woman and the other ones are photographs of the specimen.

This condition is caused by a fungus called mycetoma and can occur anywhere in the body, but most frequently occurs in the foot.

Most of the cases give a distinct history of some injury. Dr. Pagenstecher in San Antonio reported two cases in 1922. Both of them had an injury, I believe—one dropped a brick on his foot—the other one a board. They nearly always form sinuses, and these nodules break down and discharge. There are three main types, according to the character of the granules that are expelled from the sinuses. The disease goes along slowly until it involves the bones. It does not metastasize either through the blood or the lymph channels, but grows by direct extension. You can see the x-ray pictures and Dr. Waite will show you the specimen. The only successful treatment is surgery.

Case Report by Dr. Hugh W. Crouse, El Paso, Texas.

Male, age 51; single; occupation, printer. Height 5 ft. 5½ in.; weight 126; lost 10 pounds; usual, 136.

Chief Complaint: Began having trouble in July or August with swelling and affected glands under left ear. Dr. Schenck thought it might be from his teeth. Had them all pulled. Started to go down and get better, but the rainy weather set in, he caught cold, and the swelling rose again; after that was over, the snow and cold weather set in, and the trouble gradually increased. Last Wednesday it broke. Dr. Schenck told someone else —not him—that he did not know whether it was tuberculous, or something else. When it opened, there was quite a bit of colorless, sticky liquid came out. Has been running off and on ever since it opened; last night ran considerably. Is now getting to be somewhat bloody looking. Very, very painful. Burning pain, and then every once in so often a sharp shooting pain up into the ear. More painful before broke. Particularly bad at night—possibly because he notices it more.

F. H.: Father died 68, stroke of paralysis.

Mother, general debility following change of life—about 60. Three brothers, one sister, good health. Others died in infancy. No tuberculosis or cancer in the family.

P. H.: Smokes. Says he has a cigarette in his mouth all the time, whether lighted or not. No illnesses until in 1907 had pneumonia—light attack. No other serious illnesses until this commenced. Shoulder broken in 1901—street car accident—no other serious accidents. No operations. Has always used liquor more or less; never in excess. Has been drunk, but has never made a habit of it. No venereal story. Had pyorrhea before the commencement of this trouble. Appetite good, ordinarily. Sleeping poorly on account of the pain. Is sleeping better now than he did before it opened. Has been taking sal hepatica every morning; if he does not, is constipated, since this trouble came on. Bladder all right.

P. E.: Pulse 126; S. B. P. 136; D. B. P. 84; P. P. 52; E. I. 27720.

Varicosed condition of veins of legs. Skin rather dry. Patient rather emaciated. Small epigastric hernia about 6 centimeters above navel. Joints seemingly normal, outside of ordinary changes that labor would produce. On left hand side, in the region of the carotid, radiating back into the mastoidal area of the carotid, is a decidedly enlarged tumor, which runs on down practically to the middle of the neck, quite cleanly demarked; extends back pretty nearly to the middle occipit. Undoubtedly a carotid tumor. Nature of such to be decided after blood has been gone over, secretions have been examined, and general history of patient weighed.

Teeth have all been extracted.

Tongue coated, rather darkened, brown, slightly tremulous. Tongue decidedly thickened.

Tonsils enlarged; left side seems to be involved along with the carotid growth.

Carotid growth has been described. Glands, supra-clavicular, infra-clavicular, both sides, not involved. Subscapular glands are not involved. Axillary also free of any involvement. Epitrochlear glands, mammary glands, negative. Inguinal glands also not enlarged, except a little bit on the right. No signs of hernia.

Heart, apex beat two centimeters below, about one inside of left nipple. All valve sounds, while rather faint, particularly over the pulmonary and aortic, no irregularity or suggestion of any valvular trouble.

Lungs: Left, there is a lagging in the respiration, but no distinct roughness. Right, free of lagging and roughness. Posteriorly, there are some coarse rales at the apex of the left; extends down to the root. Pretty nearly vesicular in their nature. Base of lung clear. Right, free of any rales or roughness. This chest will be examined with the x-ray—stereo pictures and fluoroscopy to be made. Reasons for investigation with x-ray, the fact of the growth immediate vicinity of the luug might be a metastasis into the lung.

Liver seemingly normal in size. Gallbladder negative to Murphy test. Stomach; quite a fair Rovsing. Abdomen pretty nearly scaphoid. Percussion of stomach shows greater curvature about a centimeter below the navel. General percussion indicates there is some dilatation of this stomach. Kidneys; palpation shows right kidney decidedly enlarged. It is movable and either congenital or secondary enlargement. Left also palpable, slightly enlarged, but the right is decidedly enlarged. Appendix; Murphy test, negative; deep percussion negative.

Neurological; there is an irregularity of the pupils. Right larger than left. Both these pupils react to light. Reflexes, I would classify as normal.

Blood; Wassermann negative; Noguchi negative.

X-ray; Fluoroscopy shows apices black, anteriorly; absolutely no light in them. Posteriorly, some change under cough. Postbronchial space cloudy. Diaphragmatic angle fair. Diaphragmatic excursion poor. Like it is held by adhesions in the center. No fluid seen.

Stereorodiograph shows an odd buttoning out of the arch of the aorta just to the right of the mediastinum; at first glance seems like it is the aorta, but more careful study seems to indicate in all probability it is a calcareous degenerated peribronchial gland. This is in immediate proximity to the sternal end of the fourth rib, and extends up to the third. The heart does not go beyond the mediastinum to the right. In other words, its shadow seems to be concealed by the sternum itself, demonstrating this is a very small and pendulous heart, Roots of the lungs both thickened, striated, and shot with old, mild, years-ago tubercular process, healed. Apices of the right lung shows old tubercular condition. No activity is demonstrable, although there has been considerable involvement, as low down as the top of the fourth rib. Hyalin lines run down to the diaphragm on the right side, demonstrating two adhesions to the diaphragm, one a very acute angulation of the diaphragm. This lung is shot clear through with some striated points, particularly around its root. Diaphragm on the left is in very fine shape indeed; some thickening around the peribronchial gland; left apices a little bit hazy; would seem to have been considerably old, healed tuberculous process, the main process having been in the right lung, and some involvement in the peribronchial gland left. No signs of metastasis or malignancy.

Diagnosis: Malignancy of left parotid gland.

Operation was the combined technic of dealing with presumable malignancy of the parotid, left side. Incision was Y-shaped; one incision starting over the zygomatic process, sweeping down and then about the middle of the neck joining with the posterior leg of the Y, which extended from the upper

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Roentgenologist

EDWIN D. WARD, M.D.
Assistant Medical Director

T. C. CROWELL, M.D.
Pathologist

portion of the mastoid to unite with this. Then a straight line pretty nearly to the clavicle. The two legs of the Y were united by sweeping around close to the ear. All structures were carefully isolated. The facial nerve was located at upper lobe of parotid, which did not seem to be involved. Therefore, three branches of the facial and careful dissection with scissors and gauze was carried out to avoid injury to this important nerve. The lobe was dissected off carefully, and then the incision from below gradually lifted up so as to do a good deal like the Colonel Gray debridement technic in infectious or block removal of Crile of all glands. The lower area of the wound was then followed up posteriorly, anteriorly and inferiorly, and the tumor, which extended deep into the deeper structures of the neck, posteriorly and anteriorly, were separated from the nerve supply.

The spinal accessory was sought and located. The various branches of the cervical were located also. The sterno-cleido-mastoid muscle was incorporated in the tumor, and had to be resected. The diagastric also. The tumor lay on the internal carotid. The superficial and the deep jugular were exposed and had to be ligated. The entire mass was slowly dissected out, at times with the cautery blade, where important vessels and nerves did not forbid, and the supra-clavicular fat removed by cautery, and the tumor gradually lifted out. The tumor was a caseated structure. It extruded the substance both anteriorly and posteriorly.

Length of operation was three and one-half hours. The mass was finally and eventually lifted out in toto and measured 14 centimeters longitudinally and 12 centimeters transversely. The thickness of it was 8 centimeters, being an ovoid. Condition of the patient varied on the table; at times pulse being rather irregular; highest rate was 120. Patient left the table with pulse 100, rather cyanosed. A careful hemostatic technic was carried out. There was no loss of blood. Operation was painstakingly slow and careful. Returned to bed with pulse 100. Given three pints of soda dextrose, usual rectal administration, given in the first forty minutes; 1000 ccs. of hypodermoclysis postaxillary line injections given.

An hour and a half after returning to bed, patient suddenly collapsed and died. It was believed to be due to cerebral embolus account of the base of the brain being so closely approximated with the growth. It was intended that the wide space where the skin could not be approximated should be closed later, Wulff skin and fat graft method, the wound to be left open for better entrance of deep x-ray therapy.

A postmortem was held by Dr. Willis W. Waite on account of the fact that the public health officer is exceedingly careful as to reporting causes of sudden death, and Dr. Wilson, the health officer, demanded that postmortem be held before he would issue a death certificate.

Note: What was pathologist's report on tumor removed?—Ed.

Report of Postmortem.

Body embalmed before examining. Body that of a slender man apparently somewhat past middle age, about moderate height. No distention of abdomen, no enlarged glands. On left side just below ear, extending down into the neck, there is a large wound, partially closed but partly open, exposing structures beneath. This has the appearance of being very recent. Skull was of moderate thickness; brain was removed and completely sectioned and examined, but revealed no gross pathological changes. Median incision made from top of sternum to pubis. Small amount of subcutaneous fat, muscles rather poorly developed. On opening the abdomen no excess of fluid. Omentum free and contained scarcely any fat. Intestines practically empty and contracted, free from gas; no adhesions noted anywhere in the abdomen. Mesentery, both large and small bowel, was rather long. On opening chest, right pleural cavity was obliterated with fibrous adhesions, but the lung could be separated from the pleura without tearing the surface of the lung. The left lung was collapsed and quite markedly contracted. No excess of fluid in pleural cavity. A few hard nodules at the apex of the left lung. There were a number of rather long dense fibrous adhesions between the lung and the wall of the cavity. Right lung, on removal, is quite solid and firm in most of the upper half of the upper lobe, the rest of the lung air containing. On section, two-thirds of the upper lobe contained numerous rather deep pigmented dark gray areas. In one or two places there are caseous nodules, slightly larger than peas. The rest of the lung is more or less pigmented and contains some necrosed fibrous tissue. Left lung on removal is quite collapsed, but still contains some air. On section, grayish pink color; on pressure, small amount of bloody fluid exudes. Pericardium contains no excess of fluid; heart small and contracted. On removal tricuspid ring barely admits two fingers; mitral ring admits only the large finger. On section, all the valves are intact, but the flaps of the mitral valve are somewhat thickened and contain patches of sclerosis. The aortic cusps are somewhat thickened and contain patches of sclerosis. The aortic cusps are somewhat thickened and there is a slight amount of sclerosis at the base of the aorta. Kidneys on removal are of pinkish color and on section striations are fairly well made out. Liver, about normal size; over right lobe is considerable reddish discoloration. On section, there is a mottled appearance, the dark red areas intermixed with areas of a lighter brown color. The darker areas seem to be a little softer than the areas that look more like normal liver tissue. This change is confined to the right lobe. Gallbladder moderately distended with bile, free from stones.

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Diagnosis: Spontaneous pneumothorax, fibrous pleurisy of the left lung completely obliterating; chronic fibrous tuberculosis, apices both lungs, most extensive in the right; fibrous pleurisy of the right lung.

Dr. Waite gave a short talk on diphtheria, stating that in cases of children with scabby noses, culture was usually positive.

No further business appearing, the meeting was adjourned at 10 p. m.

CHAVES COUNTY MEDICAL SOCIETY

Dr. C. M. Yater, Secretary

At the regular meeting held on the night of February 26th, a very interesting case was reported by Dr. H. A. Ingalls, as follows:

Mrs. C., age 29 years; family history unimportant.

P. H.: Had the usual diseases of childhood. Married at 18; mother of two children (10 and 4). First labor normal. At second confinement within 24 hours began having eclamptic seizures which lasted intermittently over a period of 12 hours, from which she made a good recovery. There were no abnormal urinary findings during either pregnancy. Seven months ago she became pregnant the third time. During this last pregnancy weekly urinalyses were made which showed, at times, a few narrow hyaline casts, no albumen or blood, but much mucus, crystals and epithelial cells; blood pressure, systolic 137.

P. I.: About three weeks ago patient felt numb, with distress in the lower part of right chest. There was no loss of consciousness nor was there muscle spasm; she soon recovered. One week later she had a similar attack, which also passed off as the other. Five days after the last attack she experienced another and similar attack, coming on soon after breakfast. Following this attack the urine showed a trace of albumen, with other findings as above stated. During this attack, in the absence of Dr. Ingalls, Dr. Fisher was called and administered five minims of Norwood's tincture. Twenty-four hours later the patient was removed to St. Mary's Hospital for observation and treatment.

On admittance, T. 98.6, P. 70 and regular.

Orders: Calomel purge. Shortly after one o'clock the next morning hospital reported patient in convulsions. In consultation with Drs. Fisher and Phillips it was deemed imperative that gestation be terminated at once. Under chloroform the fetus and placenta were delivered at the cost of quite an extensive laceration of the cervix, but no perineal tear. Cervix repaired and patient appeared to be in apparently good condition. The placenta showed three areas of degeneration. It is quite remarkable that all through the disease the pulse remained slow and regular. Following delivery the urine did not

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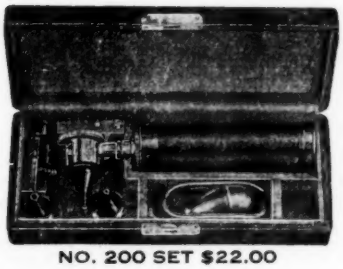

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show any evidence of acute renal condition.

On second day after delivery patient developed a deep hematogenous jaundice, but expressed herself as "feeling very well", and desirous of being returned home, as her children were ill, and she did not like hospital life.

About five o'clock that afternoon she conversed rationally with her nurse and said she was feeling well. A short time later the nurse found she had become irrational, and in the absence of Dr. Ingalls, Dr. Fisher was called in from another room in the hospital and gave a hypodermic of ten minims of Norwood's tincture and notified Dr. Ingalls. A sweat was ordered and on the arrival of Dr. Ingalls at the hospital he was informed that the patient had been quiet, with no spasm of any kind. The pulse was soft and regular and pupils dilated, skin dry and patient unconscious.

In about ten minutes she went into a brief convulsize seizure. The sweat bath not having produced the desired effect, a hypodermic of pilocarpine was given, which was soon followed by moderate sweating. At no time was there a weak or rapid pulse. About an hour before death she became nauseated and vomited a considerable quantity of blood.

The normal liver dullness disappeared during the latter hours of life. Dr. Ingalls was uncertain whether the atrophy of the liver was primary or secondary, as yellow atrophy often occurs in the non-pregnant state as well as in the pregnant, and was of the opinion that the underlying cause in this case must be considered as unknown.

The hour being late, the discussion was very brief, all agreeing with Dr. Ingalls' conclusions.

CHAVES COUNTY (N. M.) MEDICAL SOCIETY

At a regular meeting of the Chaves County Medical Society held in Roswell, N. M., February 12, 1924, the following resolution was unanimously adopted:

Whereas;

(1) Under the Harrison Narcotic Act of 1918, an excessive and now unnecessary war tax is imposed on the medical profession, and

(2) The physician is compelled to pay an income tax on money paid out by him for certain expenses of his profession and is thus taxed on an amount in excess of his net income, and

(3) The physician is now taxed on his earned income at the same rate that he and taxpayers generally are taxed on incomes from investments, speculations, etc.;

Therefore, be it resolved by the Chaves County (New Mexico) Medical Society:

First: This society desires it to be distinctly understood that we are not opposed to the Harrison Narcotic Act itself, nor do we oppose any rate of tax under said act that may be necessary to adequately enforce the same, but we do protest that any tax in excess of the minimum amount necessary to enforce said act is an occupation tax on the physiian and discriminates against the medical profession, seeing federal occupation taxes are not imposed on other professions; and when passed on by the physician to his patients is a tax on the sick and injured.

Inasmuch as this law was enacted for the benefit of the community, and not for the benefit of the medical profession, there is no reason for imposing on the medical profession any greater part of the cost of enforcement than is imposed on any other group in the community.

Second: This society protests the ruling of the Commissioner of Internal Revenue that **"a physician who is away from home in attendance at a meeting of a medical society or while pursuing postgraduate studies is not away from home in the pursuit of his profession, and that the expenses incident to such travel and studies are not ordinary and necessary expenses of the practice of medicine."**

This ruling ignores the fact that such expenses are absolutely necessary in order that the medical profession may be better equipped to scientifically treat the sick and injured, and should be exempt from taxation.

This ruling is inconsistent with the provisions of the law generally as they relate to the practice of medicine.

For instance, a physician may deduct medical society dues paid, but he must pay tax on money expended in attendance on meetings of the same society.

This ruling is further inconsistent, in that, if a physician travels from one place to another to consult with another physician regarding the treatment of a single patient he can deduct traveling expenses; but if he travels between the same two places to consult with a hundred physicians about the best mode of treatment of sickness generally, he is not allowed to deduct expenses.

If a physician travels from one place to another to examine one patient, in order to apply his skill, for the benefit of that one patient, his traveling expenses are deductible; but if he travels from one place to another to engage in postgraduate study in order to make the knowledge and skill thus acquired available to the entire community which he serves, he is not allowed to deduct traveling expenses, but must pay income tax on them.

This ruling tends to discourage postgraduate study and meetings of medical societies and is indeed very poor public policy; inasmuch as medical society meetings and postgraduate study tend to conserve public health.

Such meetings tend to bring about a more patriotic unity, and instead of being taxed should be encouraged.

Third: That this society heartily concurs in the provision of the proposed revenue law making the rate of taxation on earned in-

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(1) Z. Zsigmondy—Anal. Chem. 40 (190), 697; Beitr. Physiol. Path. Chem. No. 3 (1903), 137.
(2) Drs. Moore and Krombholz, J. Physiol., 22 (1908), 54.
(3) Dr. C. A. Herter ("Infantilism from Chronic Intestinal Infection").
(4) Dr. Abraham Jacobi ("The Intestinal Diseases of Infancy and Early Childhood").

comes less than the rate of incomes from investments, speculations, etc.

Such taxation is a tax on the productive activity of the individual and tends to discourage such activity; and since the productivity of the individual may be destroyed at any time, by sickness, or accident, and is surely destroyed by death, it should be entitled to special consideration when determining the rate of taxation applicable to such income.

Be It Further Resolved, That copies of this resolution be filed with the Committee on Ways and Means of the House of Representatives, the Committee on Finance of the Senate, and each of our Senators and Representatives in Congress.

C. M. YATER, Secretary.

Dr. A. A. McDaniel of Lovington was a visitor in Roswell the first week in March.

Dr. Bayard Sullivan of Fort Bayard, N. M., has been elected secretary of the Grant County Medical Society, to fill out the unexpired term of Dr. H. S. Bennett, deceased.

BERNALILLO COUNTY MEDICAL SOCIETY

Albuquerque, N. M.

The regular meeting of the Bernalillo County Medical Society was held Wednesday evening, February 6th. The new president, Dr. David C. Dodds, gave the society a talk, explaining his aims and ambitions for this year. If Dr. Dodds is able to carry out his plans, the society is going to be one that the members cannot afford to miss.

A committee of three were elected whose duty it is to hear and adjust all grievances between members during the year. This committee is composed of Drs. P. G. Cornish, Sr., L. G. Rice and J. A. Reidy.

It was suggested that the society had as members in good standing a few men that should not be members, and it is refreshing to think that someone in the society may start something that will raise the standard.

The subject of newspaper advertising was taken up and it was decided to leave this phase of things up to the above named committee. The state society already has passed a resolution, the gist of it being that newspaper advertising disqualifies a physician for membership in the state society. This no doubt will be the attitude of the committee acting upon local affairs.

MARICOPA COUNTY MEDICAL SOCIETY

(Phoenix, Arizona)

February 2nd Meeting

Held at the Deaconess Hospital, Dr. Charles S. Vivian, president, presiding.

Communication from the Custer County, Okla., Medical Society, transferring Dr. T. T. Clohessy of Buckeye, was ordered referred to the Board of Censors for their approval.

President read a letter from the Medico-Legal Budeau of the A. M. A., addressed to the chairman of the Committee on Public Policy and Legislation. It was suggested that a committee be appointed to draft resolutions in accordance with the suggestions of this letter, to be forwarded as the expression of the society. The committee appointed were Dr. H. R. Carson (chairman), Dr. W. W. Watkins and Dr. Win Wylie.

Announcement was made by Dr. D. F. Harbridge regarding the public meetings in the interest of public health. There are to be meetings in Phoenix, Tucson, Globe and Nogales, in an effort to place before the people the value of legitimate medical practice. It was moved and carried that the society's Committee on Public Policy be appointed, and this matter placed in their hands, with instructions to co-operate with Dr. Harbridge. The committee appointed was Dr. Fred Holmes (chairman), Dr. L. H. Thayer and Dr. Mary Neff.

Time was given Mr. Duffy, an attorney, to explain the situation with regard to prescribing narcotics in this county, and certain dangers which the interpretation of the law by the Federal Court of this district introduces. These dangers came out in the trial of Dr. Aiton, who was convicted on one count of the charges against him for prescribing narcotics illegally. Mr. Duffy's statement was to the effect that conviction in this case opened the way for any doctor in Maricopa County who prescribes more than the usual amount of a narcotic for a person who is not an addict to be indicted under this interpretation of the Harrison Act. The matter was ordered referred to the Committee on Public Policy and Legislation just appointed.

The society then passed to the consideration of two clinical cases, selected from "Cabot's Histories."

The first case was discussed by Dr. Goodrich. The case was that of a seven-year-old girl with paralysis of both legs from old infantile paralysis. A week before she had slipped and fell on a needle which she was carrying in her right hand, puncturing the skin over the right clavicle; the needle apparently made only a superficial puncture. Two days later a small swelling developed at site of injury, which gradually increased in size and tenderness, producing finally a rounded, fluctuating, red and tender swelling. T. 202; pulse 150, leucocytes 26,000. X-ray showed collapse of right lung, with some fluid and air in the soft tissues over the upper part of chest. Operation was performed, but child died next day.

Dr. Goodrich considered the child received in the puncture wound some virulent type of infection. and the principe interest would seem to center around the type of infection. If we do not regard the injury as sufficient to puncture the lung, we are forced back on gas bacillus infection to explain the air in the tissues. There are four or five types

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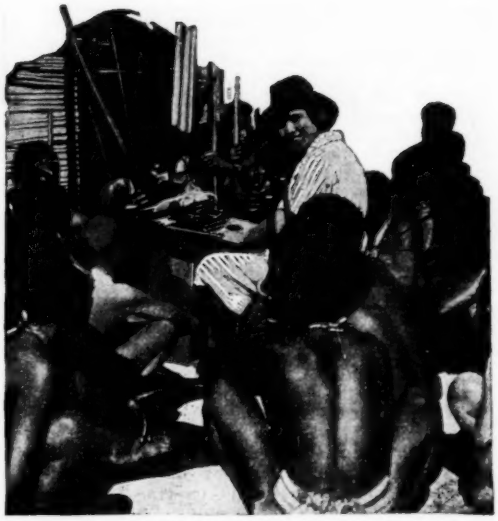
Travel—adventure—danger—hair-breadth escapes! The stories Corona has told—the sights it has seen! The deadly African jungle, the Chinese bandits, the head-hunters of the South Seas, the huddled ice huts of the Arctic.



Mrs. Martin Johnson, wife of the famous wild-animal photographer, seems quite at ease as she demonstrates Corona to a group of head-hunting savages. Mr. Johnson has carried this Corona twice around the world and says it works as well today as the day he bought it.

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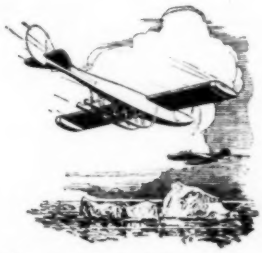

There are no typewriter repair shops in the Arctic, but that doesn't worry Captain Roald Amundsen who will carry Corona on his trans-polar flight. Mr. H. H. Hammer, his personal representative, says, "Corona has been chosen particularly on account of its thoroughly tested and proved durability."

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of bacteria which produce gas; B. of malignant edema; B. aerogenes capsulatus of Welch, colon bacillus, proteus and a psuedo type of B. malignant edema. We can rule out all but two of these, since neither the colon, proteus nor the psuedo type of malignant edema would cause such virulent infection; that leaves malignant edema and aerogenes capsulatus, and many features of the case make us think of malignant edema infection. It is found in the dirt on the floor of houses. If it was this infection, cannot quite understand the expectant treatment for 24 hours. The chest findings could be explained by the infection, with lung collapse. The ease with which the needle was extracted makes us think it did not enter the lung.

Dr. Holmes thinks can hardly count on a history in a child of seven years. This needle could easily have punctured the subclavian vein and entered the pleura and apex of lung. Tme swelling is not the swelling of emphysema, but fluid; thinks that puncture of the vein with slow leak of blood into the tissues, with lung puncture causing the lung collapse, with infection coming later, would explain all the symptoms. Air in the tissues could come from the punctured lung. Does not think that gas bacillus infection quite covers, without lung rupture.

Dr. Goodrich thinks that if the lung is punctured will have emphysema at once; this child sticks needle into the skin and 48 hours later a small swelling appears, and severe symptoms do not appear until nearly a week after the injury.

Dr. Brown thinks that infection might have been carried in and the lung punctured at the same time.

Dr. Mills thinks that gas bacillus infection would have produced gas in the tissues earlier.

Dr. Phillips thinks that a very superficial wound might puncture the lung above the clavicle in an emaciated child; nothing in the case that could not be explained by partial pneumothorax and infection.

The operation performed in this case was a thoracentesis and removal of very foul bloody fluid.

Necropsy showed gangrene of the superior lobe of right lung with atelectasis, and gangrene of chest wall in region of first rib, of the tissue bordering the right clavicle and of the mediastinum extending into the neck. There was no bacteriological report, and it is not impossible that there was a gas bacillus infection, though this was not proven one way or the other.

The second case was discussed by Dr. Gudgel. It was that of a boy, age 11, with shortness of breath and dyspnea, together with swelling of feet and legs. For three years had been having attacks of pains in knees and legs. Six weeks before admission had sore throat and fever; week before admission tonsillectomy was advised because of large, swollen tonsils and infected cervical

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glands.

P. E.: A well-nourished boy with cold and cyanotic skin and an anxious expression, in very great respiratory distress, unable to lie down and on the least exertion becoming cyanosed and prostrated. Chest expansion decreased on both sides. Lungs: Impaired resonance, with moist and sticky fine rales in both bases back and front and in the axillae. Breath sounds over both bases bronchial and markedly diminished in lower anterior right chest. Apex impulse of heart felt outside the nipple line, not particularly forcible. Dullness 5½ inches to the left, 4 to the right. Sounds somewhat distant. A slight systolic murmur heard only outside and just below the left nipple. Slight thrill over precordia felt just to left of sternum. Liver border on level with umbilicus. Marked tenderness over liver. Extremities: Both feet and ankles and region over spine in lumbar region markedly edematous.

T. 98.5°—105. P. 126—156. R. 39—60. B. P. not recorded. Urine: Amount 30 on the one occasion recorded. Sp. gr. 1024—1018. Smoky at one of two examinations, large trace to a trace of albumin at both, full of leucocytes and red blood corpuscles at both, rare granldar casts at the first, many waxy and cellular at the second. Blood: Hgb. 80%—85%. Leucocytes 35,800—36,000. Polynuclears 86%. Reds and platelets normal. Wassermann strongly positive. Non-protein nitrogen 110 mgm. Creatinin 197 mgm.

Patient was put on stimulant treatment. Pericardium was tapped and sterile fluid withdrawn, with marked temporary relief. Fluid appeared in pleural spaces and patient gradually failed and died.

Dr. Gudgel thinks the symptoms point to the heart, although pneumonia must be ruled out. The respiratory difficulty might come from simple dilatation, but there would seem to be a more serious condition than simple dilatation; the increased size and systolic thrill usually means some valvular lesion. The presence of fluid in the pericardium naturally means pericarditis. Endocarditis and pericarditis would explain all the symptoms, dyspnea, passive congestion of lungs and liver; urinary findings could result from this also.

Dr. Watkins thinks the urinary findings plus the non-protein nitrogen must mean nephritis.

Dr. Carson thinks the tonsil infection has produced nephritis.

Dr. Smith thinks that if we could go back two generations and eliminate syphilis, then take out the tonsils and treat nephritis, the patient might have been cured.

Dr. Phillips thinks syphilis would explain the symptoms.

Dr. Holmes recalled a case seen at the Massachusetts General Hospital, where they brought in a patient to have tonsils removed and there was a large chancre on one. If this patient had an acquired syphilis, it would

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1909	105,000

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be about the time for secondaries to occur.

Dr. Brown thinks the deep cyanosis is rather characteristic of pericarditis with effusion; a streptococcus infection in the tonsils going down and infecting the lungs; then becoming systemic, involving heart, pericardium, kidneys and blood, would cover the case.

Dr. Mills thinks the effusion in the pericardium is part of a general anasarca; the feet, ankles and lumbar region were all edematous. Believes the sequence is a streptococcus infection producing pericarditis and along with it an acute nephritis with general edema.

Dr. Goodrich said he has seen a replica of this case, caused by general gonococcus infection, with pericarditis, fluid in chest and nephritis.

Necropsy in this case showed pneumococcus septicemia, subacute pericarditis, serofibrinopurulent pleuritis, hypertrophy and dilatation of the heart, chronic passive congestion. Heart valves were not diseased, layers of pericardium were bound together, the hypertrophy resulting from overwork of heart; microscopic examination of kidney showed acute glomerulonephritis, although gross inspection could not determine whether nephritis was present or not.

Society adjourned at ten o'clock.

ST. JOSEPH'S HOSPITAL STAFF MEETING

Phoenix, Arizona

The staff of St. Joseph's Hospital, Phoenix, Arizona, held its regular monthly meeting on February 7th.

The first case on the program was one in which the staff members were invited to make a diagnosis, a box of cigars being offered to the member who would forecast the autopsy findings.

The case was that of a man, married, age 42, machinist by trade.

His wife died of tuberculosis; had diphtheria 15 years ago and gonorrhea 25 years ago.

Six months ago had a sharp localized epigastric pain and much nausea after supper, gradually disappearing after three to four hours' rest and application of heat. Has had other attacks since of dull pain in epigastrium with nausea and sense of fullness of stomach every two or three weeks, lasting four or five days. Has vomited occasionally. Four months ago had loose, watery movements for two months. Five weeks ago had sharp attack of pain in epigastrium which shifted from side to side; since then has had dull ache and sense of fullness in epigastrium, with nausea. Two weeks ago, after some irrigation in throat, raised a quantity of bright red blood with foam; has been spitting up dark brown blood since; appetite poor; weight 120; used to be 150.

P. E.: Pyorrhea. Fine crackles, with sibilant and coarse rales over upper right chest anteriorly; left chest hyper-resonant.

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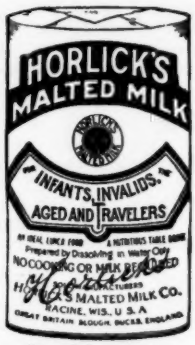

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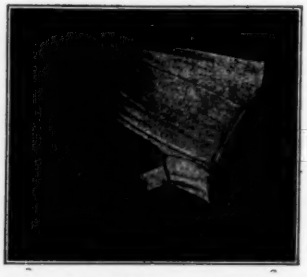

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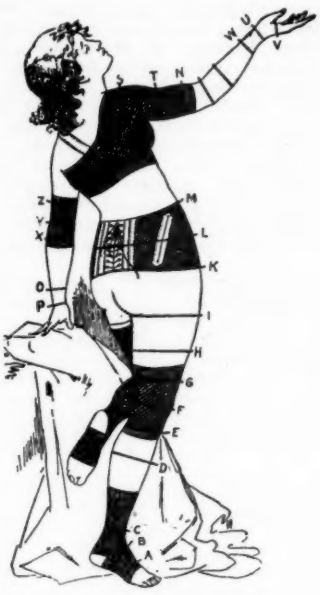

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Suggestions that point out the procedure to be followed in adjusting the diet to overcome constipation due to the stated causes are embodied in a 16-page pamphlet, which will be sent to physicians upon request. The suggestions offered are based upon careful observation extending over a long period and should be of much service to every physician who is at all interested in infant feeding.

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Fine and coarse rales behind in left base; sibilant rales in right base; no signs of consolidation or cavity. Apex beat in sixth space 10 cm. to left; heart rapid, sounds poor in quality; loud systolic murmur over entire precordia; harsh at base; questionable diastolic at pulmonic. B. P. 100/90.

Abdomen tender; liver edge palpable. Fingers clubbed.

Sputum; neg. for T. B. Urine trace of albumen, granular and hyaline casts. Leucocytes, 4,400, 20,800 and 8,800. Wassermann negative; two blood cultures negative.

X-ray: Heart enlarged to left. No chest shadows characteristic of tuberculosis. Barium meal shows crater defect in duodenum, with six-hour residue. Appendix region not examined.

Several diagnoses were made—tuberculosis, heart disease, gallstones, duodenal ulcer, cirrhosis of liver.

The doctor who handled the case not now being in Phoenix, Dr. Watkins discussed the case.

The history of the case would suggest an abdominal lesion—ulcer, gallbladder, kidney stone or appendicitis. Hemorrhage would bring the possibility of tuberculosis into the foreground.

The physical examination centers the attention on the chest and heart; the rales and history of exposure, with hemorrhage would be sufficient for many doctors, but the predominance of basal signs would make one pause before diagnosing tuberculosis. There is evidently a heart lesion, not because of the murmur, but because of the enlargement. Clubbed fingers have a significance, and point to lung or heart disease.

Laboratory findings do not add a great deal, except the negative sputum and the negative x-ray findings would, for me, rule out tuberculosis. The barium meal would seem to clinch the diagnosis of upper abdominal lesion, probably duodenal ulcer.

Final diagnosis would be duodenal ulcer and organic heart disease—probably in mitral valve.

Patient grew progressively more dyspneic, in spite of stimulation, and finally died with symptoms of heart decompensation.

Autopsy showed the heart enlarged, chronic and acute endocarditis with vegetations on the mitral and aortic valves. Lungs, no tuberculosis, with numerous very small infarcts. There was an old adherent pleura on right side. Liver passive congestion; kidneys normal; spleen with infarcts. Stomach and duodenum without evidences of disease. No ulcer present. Chronic appendicitis, with adhesions and concretions.

No member won the cigars.

Case 2 was presented by Dr. Felch. It was case of a man, aged 64, who, three weeks before, had suffered fracture of the radius.

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Repeated efforts to hold it in position failed, and union had not begun at end of three weeks. Failure to hold bones in position and seeming delay in union led to operation of Lane plating. At the present time bones are still in good position, but there is no sign of callus as yet shown by x-ray. No particular reason for delayed union, except his age and possible syphilis.

Dr. Beauchamp states that surgical books tell us that these cases should not be plated short of six months or a year.

Dr. Carson said that Murphy used to teach that keeping the ends immobile did not assist callus formation; recalls a man 60 years old who had a fracture which would not unite and used to rub the ends of the bones together and use hyperemia. Blood can be injected between the ends of the bones.

Dr. Watkins stated that there have been some interesting observations on the effects of high voltage x-ray treatments and the effect of these on bone formation. It is well worth trying in such cases.

Dr. Sweek thinks it bad practice to do open operation on any fracture when there is possibility of getting results without this. Sometimes callus is not laid down for many months, and fractures of leg or forearm should be given several months and every other means exhausted to get the bones to unite before using open operation. Only when you see that the union is going to be vicious and the functional result bad is open operation called for. In the open operation, it is better not to introduce a foreign body.

Dr. McIntyre: According to the Mayos, a fracture is not regarded as a non-union until two or three years have elapsed; until then, it is regarded as delayed union.

DR. Felch: Patient persisted in getting the bone out of position, and operation was done to hold bone in place and cut down time in the hospital.

Dr. Wylie recalled a case of fractured humerus with perfect apposition which, at the end of three months, had not grown callus. Put in a bone splint across the fracture; after a month, x-ray showed a liatle callus on one side; after another month perhaps a trifle more; after six or eight months no further increase in callus. While in Los Angeles, received telegram from attorneys of a meeting next day. Came back to prevent settlement, and found they had settled for $4,500. Some doctors had told him his arm was worthless; he had demonstrated absolutely that he would not grow bone. One year later, that man was working regularly as a mucker and his arm is just as good as it ever was. Would not grow bone, but did, time being the only thing necessary.

Dr. Felch stated that he did not approve of plating everything that comes. Now and then plates one. Does not think the plate will interfere with callus formation.

Dr. McLoone presented the third case, the history of which was as follows:

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Merchant, age 35; mother and father living and well; married and has three children.

In March, 1923, had a severe head cold, following the acute stage of which there was a chronic nasal discharge. Post-nasal dropping and frequent sore throat. Following a severe attack of rhinitis began to have epilepsy, attacks occurring once in two weeks until they were made less frequent by treatment which was started in May; discontinued treatment in July and had another attack after three or four days. Went to Silver City in April and Dr. Bullock examined his lungs; found negative. Tonsils removed in May. About middle of August began to have pain in left ear; drum ruptured and discharge lasted for two weeks, then ceased. Three weeks later, in early part of September, right ear began to pain; says pain was almost constant and describes it as being deep in ear. Pain was still present when seen on October 14, 1923.

P. E.: Ears, whisper, A. D. 8 ft. A. D. C2—A"5—B 16". A. S. 15 ft. A. S. C2—A 20" 10".

Weber lateralized to right.

Right drum retracted and thickened; cone of light diminished. Some retraction of left drum head, with adhesions. Result of tonsillectomy, good.

Sinuses: There is a marked deviation of septum to left; no pus; on right, some pus

on ant. end of middle turbinate. Pus coming from sphenoethmoid recess; by washing right antrum, one-half ounce of sero-sanguineous discharge obtained.

Right ear canal was swollen and external otitis was treated; this subsided in two days, but pain continued. Drainage of right antrum and sphenoethmoid regions gave no relief from pain. On account of the subacute inflammatory thickening of Schrapnell's membrane, incision was made in right ear drum; no pus, only a slight amount of blood.

X-ray: All right sinuses showed a slight relative increase in density as compared with the left. The mastoids showed a well-developed pneumatic type of cell, but with loss of definition and increased density. X-ray would indicate low-grade infection in both mastoids.

There was no fever at this time; a latent mastoid abscess was suspected on the right, and exploratory incision advised. Patient had to return home, however, and was not seen again until November 16th; he then stated that three days after returning home his right ear began to discharge. His chief complaint now was deep seated pain about one inch below tip of mastoid, along the border of the right sternocleidomastoid muscle. Slight pulsating discharge from right ear; very little tenderness over mastoid antrum. Very little sagging external to Shrapnell's membrane. Hearing in this ear had diminished to a whisper at four feet. All the sinuses were negative, except for a slight purulent discharge from the posterior ethmoidal cells on the right.

X-ray about one month later stated that the difference in the two mastoids was not great; slightly more haziness and loss of definition on the right. Probably pathology on both sides, with more evidence of recent infection on right.

Operation November 19th. Right mastoid opened in usual manner. Pus found in antrum with granulations; granulations in tip and free pus under pressure. Lateral sinus covered with large necrotic mass; gutter cells involved. On removing granulation tissue and necrotic mass over sinus, the sinus was perforated; free bleeding from both ends. Packed with iodoform gauze and drainage established; wound closed in usual manner.

Dr. McLoone stated that the interesting feature of this case was that it illustrates that we can have very severe involvement of the mastoid with very few symptoms. This man had a perisinus abscess following mastoiditis; had practically no fever; no leucocytosis, very little tenderness; on going in found very little pathology in the mastoid cells, but an abscess around the sinus.

Have recently had a second case of perisinus abscess. This was a woman who had pain in the ear complicated by external otitis, tenderness very deep, some fever, no sagging of the wall, no signs of pus under pressure. On opening, cortex was dense, practically nothing in the antrum. Decided not to enter the antrum and evacuated one or two drachms of pus from around the sinus. Took culture from the antrum and found it negative for organisms. Acute infection had become chronic, was walled off both anteriorly and posteriorly, with little symptoms of mastoiditis except over the tip. Without operation she would have had thrombosis. The x-ray showed positive mastoid involvement. If there had been doubt, would have had repeated x-ray examinations. In a recent case, had three x-rays at intervals, which showed a gradual clearing without operation.

When in doubt, especially with a history dating back six or eight weeks, am well convinced of the value of the x-ray examination in determining mastoid pathology.

In the first case had a symptom which was very suggestive of trouble in the internal jugular; the lateral sinus was covered with granulations and he undoubtedly was having a phlebitis with some infection of the jugular. Otologists are taking a great deal of note of atypical mastoiditis and by operating these cases early we avoid lateral sinus thrombosis. This patient should have been operated in August, because he was positively septic then.

Dr. Fahlen inquired whether transillumination would be of value in such cases.

Dr. McLoone answered that it is very satis-

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factory in mastoid work. Eburnation of the cells immediately around the canal would give a dark shadow and be very misleading. The x-ray is far superior. In the second case, the x-ray report showed diseased cells posterior and high up in the process, and this was borne out by the operation. Another very important point in the x-ray examination is to determine the position of the lateral sinus, whether it is forward or back. Operation on the mastoid is justifiable if you have at least one of the cardinal symptoms of mastoiditis.

Dr. Fahlen: In one case you stated the pulse was 70. At any period was the pulse slowed up by meningeal involvement?

Dr. McLoone stated that in the first case the pulse ran around 70; at one time ran as low as 60; temperature ranged from 97 to normal.

Dr. Fahlen stated that if the pulse drops, it would be a rather significant symptom, though not one to be waited for.

Dr. McLoone stated that, as a rule, as in the second case, the pulse and temperature are up; they are inclined to have the septic type of infection, rapid pulse and some fever, which the first case did not have. In both cases the classical signs of mastoiditis were absent, but still with dangerous pathology in the mastoid. If you have a slow pulse, you would begin to think of lateral sinus abscess; you have ruled out sinus thrombosis, because there is no fever, no chill, no rise in temperature, no leucocytosis, and negative blood culture. Had no reason to suspect lateral sinus thrombosis in the first case, until he did not do well after the operation; then began to suspect possible brain abscess, or atypical sinus thrombosis because of the tenderness along the sternocleidomastoid muscle, which is a rare symptom, but a possible one.

Dr. Neff asked how to explain the epileptic seizures. Answer, that they were hard to explain; occurred first after an attack of influenza; while in the hospital received no treatment for epilepsy and had no seizures until about six weeks following operation. Did not hope to cure the epilepsy by operation, because we know that when a patient is operated on for anything, his epilepsy frequently ceases for a time due to the psychological effect of the operation.

Dr. Neff inquired about accidental severing of the facial nerve in mastoid operations. Dr. McLoone replied that this could occur at the hands of the very best men, when there is destruction along the course of the nerve involving the nerve canal. There is also at times an abnormal course of the nerve.

Dr. Schwartz was on the program to present some cases of foreign body in the globe and describe method of extraction, and method of handling bodies which could not be extracted. Dr. Schwartz was not present, and this was postponed until the next meeting.

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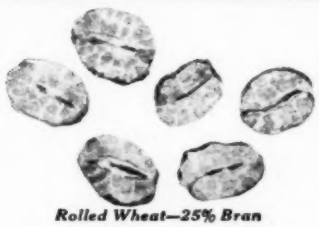

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STAFF MEETING ARIZONA DEACONESS HOSPITAL

February 23, 1924

At the regular meeting of the Medical and Surgical Staff of the Deaconess Hospital, held February 23, the following members were present: Drs. Thomas, Watkins, Mills, McIntyre, Couch, Felch, Shelley, Schwartz, Bailey, Wilkinson, Goodrich, Hicks, C. B. Palmer, McCall and Goss. In the absence of Dr. Ellis because of illness, Dr. Thomas was elected chairman and presided over the meeting.

A resolution from the Medical Council protesting against the action of the Arizona State Nurses' Association, in a certain matter vital to the hospital training school, was read, and the staff voted to approve the Council's action and instructed the secretary to send a copy of the resolution to Governor Hunt, with the names of the staff accompanying it. (It is interesting to note that the Nurses' Association rescinded its action two days later).

Dr. Bailey presented an interesting case of persistent hemorrhage following sub-mucous resection in a woman just entering the menopause, which hemorrhage did not begin until seven days after operation. The bleeding was not confined to the cut surfaces, but included the whole mucous membrane of the posterior nares.

Dr. Schwartz remembered a similar case from El Paso, in which the cause of the hemorrhage was not discovered.

Dr. Felch's case report concerned a boy of ten years who entered the hospital with the diagnosis of acute appendicitis. Operation revealed many tumor masses involving the mesenteric vessels and the small gut. The abdomen was filled with a brownish colored fluid and the tumors were reddish gray in color. The masses resembled carcinomata, but were not sectioned due to their close relation to the blood vessels. After operation much distention was observed, but the wound closed in a few days.

After discussion of both papers, the staff adjourned to meet the last Saturday in March.

H. L. GOSS, Secretary.

EL PASO COUNTY NEWS ITEMS

Dr. J. W. Tappan is the proud father of another boy, Robert Lewis, born December 5, 1923.

Dr. Will P. Rogers was married to Miss Lula B. Adams December 26, 1923.

Announcements have been received of the marriage of Dr. W. A. Toland to Miss Grace B. Terrill of Boston, Texas, November 26, 1923.

Announcement is made of the marriage of Dr. Elliott C. Prentiss to Miss Ethel J. Owbridge, at the home of the bride's parents, in Santa Monica, Calif.

Dr. J. M. Treilo, formerly physician to the Mexican-Northwestern Railroad for more than two years, is now physician to the William Randolph Hearst Ranch at Babicora, Chihuahua, Mexico. Dr. Treilo spent a few days in El Paso during December.

Dr. Harvey S. Kinard has opened offices at 504 Roberts-Banner Building, for general practice.

The El Paso Masonic Hospital has, since January 1, 1924, been entered upon the list of standardized hospitals by the American College of Surgeons.

The Waite Pathological Club is a new institution for medical study recently organized and now in full sway of its functioning. Among the few rules that govern the body are, that its membership shall be limited to twelve and its members shall not be absent from more than three consecutive meetings. It is a live organization, very enthusiastic in the study of medicine, giving themselves a continual postgraduate course, as it were.

Briefly, the work they have outlined for themselves is the careful and complete history taking of their cases and, when available, autopsies are done and the pathological tissues studied in the laboratory. In this way they have a complete study of the cases from the earliest history to completion.

DR. THOMAS WATKINS of Bisbee, Ariz., was a visitor in Phoenix during February, coming for the purpose of having some eye examinations made.

DR. ALBERT KNAPP of Chicago, formerly medical director of one of the large hospitals in Dusseldorf, Germany, was a visitor in Phoenix in February, coming as the private physician of a patient who was left in Phoenix. Dr. Knapp's special line of work covers the field of the heart and lungs.

DR. WM. C. BANE of Denver, Colo., Professor of Otology and Laryngology at the University of Colorado Medical School, has been spending several weeks in Phoenix, where he has been frequenting the golf course of the Country Club.

DR. L. M. TOMPKINS, formerly located at Gilbert, Ariz., has moved to Globe, where he is associated with Dr. W. W. Horst.

DR. T. H. SLAUGHTER of Miami, Ariz., is reported to have moved from that city to Long Beach, Calif.

DR. CROSBY of Yuma, Ariz., was a visitor in Phoenix during February, coming on professional business.

DR. JOHN E. BACON of Miami, Ariz., addressed the Kiwanis Club of Phoenix, by invitation, on March 5th, the subject of his talk being "The Copper Industry of Arizona".

DR. TOLER R. WHITE of Kingman, Ariz., president of the smallest and livest medical society in the state, was a visitor in Phoenix during the latter part of February.

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LIVER FUNCTION AND LIVER FUNCTION TESTING*

CHESTER R. HAIG, Major, M. C., U. S. A., William Beaumont General Hospital, Fort Bliss, Texas.

The liver is the largest single organ in the body and one of the most important, its presence in a state of functional activity being necessary not only for a condition of health, but even for a continuation of life itself. I like to quote the following remarks of William J. Mayo in speaking of the paramount importance of the hepatic function:

"The liver was recognized, even by the ancients, as the king of organs, standing as a buffer, on the one hand, between the general circulatory system and the gastro-intestinal tract, and, on the other hand, between the general circulation and the spleen, which filters from the blood micro-organisms and toxins that it is unable to destroy, sending them to the liver through the portal circulation for destruction and detoxication. The liver detoxicates chemical poisons, such as chloroform, phosphoms and arsenic, as well as biochemical poisons produced in the body. Yet for many reasons we know little concerning the functions of early pathologic conditions of the liver. Its concealed situation prevents us from accurate means from without of early diagnosis. Its power to regenerate injured cells and form new cells is not equaled by any other organ of the body, and its enormous margin of safety prevents us from knowing just what is taking place in the earlier stages of hepatic disease, and the appalling nature of the pathologic changes when they are sufficient to enable physical demonstration, has made the liver an organ of mystery and conjecture. I venture to predict that the next few years will bring forth productive research demonstrating that the causes of many of the changes in the so-called vital organs which lead to death do not primarily originate in the nervous system, heart, lungs or kidneys, but are the result of hepatic insufficiency leading to insufficient purification of the blood, thus making possible much sound prophylaxis.

"The functions of the liver are: (1) defense against invasion of the body by micro-organisms and the detoxication of toxic products brought to the liver from the portal circulation, (2) fat metabolism, (3) protein metabolism, (4) carbohydrate metabolism, and (5) bile function."

Howell, in speaking of the liver, says:

"Its functions are manifold, but in the long run they depend upon the properties of the liver cells, which constitute the anatomical and physiological unit of the organ, although there is histological evidence that some of the endothelial cells of the capillaries of the liver have marked phagocytic properties and it is possible that their activity may play a contributory part in some of the functions of the liver, for example, in the destruction of the red corpuscles that precedes the formation of bile pigments. The liver cells are seemingly uniform in structure throughout the whole substance of the liver. The physiology of the liver cell falls naturally into two parts—one treating of the formation, composition and physiological significance of bile, and the other dealing with the metabolic changes produced in the mixed blood of the portal vein and the hepatic artery as it flows through the lobules. In this latter division the main phenomena to be studied are the formation of urea and the formation and significance of glycogén, but it cannot be doubted that the liver possesses other important metabolic functions which at present are imperfectly understood, such, for example, as its relations to the production of fibrinogen and antithrombin."

(*Read before the El Paso County Medical Society, at El Paso, Texas, Nov. 5, 1923.)
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Boucart in Gynecology and Obstetrics, Paris, speaks of puerperal infection and insufficiency of the liver. He considers the liver of primordial importance in combating infections and claims that liver functioning flags because of inadequate blood supply. The portal circulation begins and ends in capillaries and depends on changes in abdominal pressure to force along the blood. As a therapeutic measure he uses light manual vibratory massage of the region below the liver for twenty to thirty minutes.

Hess, abroad, and Napoleon, of Boston, emphasize the importance of the liver as an organ which protects the right heart from congestion, taking up a large amount of blood during venous engorgement of a decompensatory heart.

Mann and Magath of the Mayo Foundation have persisted in their experimental studies of the physiology of the liver and after studying the effect of total liver extirpation have studied the effect of pancreatectomy in conjunction with liver function in relation to carbohydrate metabolism. They say:

"Previous studies have shown that (1) a characteristic group of symptoms followed by death develops after total removal of the liver; (2) these symptoms are associated with decreasing blood sugar and various symptoms and death occur at definite blood sugar levels, (3) the injection of glucose after symptoms develop abolishes them and restores the animal to normal, and (4) if glucose is administered after hepatectomy in amounts sufficient to maintain the blood sugar level at normal or above normal, the characteristic symptoms do not develop. It has been emphasized by our previous experiments and those of other investigators that only a small portion of the normal liver is necessary for the maintenance of its function. The results of the experiments on dogs where liver, or pancreas, or both have been removed, are very definite as regards the liver. They prove conclusively that the liver is absolutely necessary for the maintenance of the blood sugar level in the hyperglycemic animal in the same manner as in the normal animal. The increase in blood sugar following pancreatectomy is dependent on the presence of the liver. Without an adequate amount of functionating liver tissue the increase in blood sugar following pancreatectomy could not occur. We have thus proved in another way that whatever may be the process of carbohydrate metabolism, the liver has a vital part in that process. The results of the experiment do not show why the loss of the pancreas should cause the rise in the blood sugar, but they do prove that the presence of the liver is necessary for this rise.

H. Rogers, in the Medical Press, Paris, speaks of the function of glycogen and of the value of sugar in operative cases. He states that glycogen is not only a reserve, but enters directly into most of the transformations (chemical) accomplished by the liver. It is an indispensable element in the liver in handling ketone products, fats, derivatives of protein, toxic substances and even micro-organisms. Glycogen is far more than a mere testimony of liver activity, wherefore the importance of an ample supply of foods from which the liver makes its glycogen. Sugar is indispensable in infections and intoxications and in all conditions tending to induce acetonemia and acidosis. The favorable action of subcutaneous or intravenous injections of a solution of glucose is due in large measure to the reinforcing by it of the functions of the liver.

Chevrier asserts that by taking 150 gm. of sugar the evening before and again the morning of the operation, anesthetics are less liable to induce liver disturbance. He follows the operation with a sugar solution by rectal drip. Among experiments he describes are some in which .25 c.mm. of an anthrax culture, injected into a peripheral vein, killed the rabbit in 38 hours, while a dose 64 times larger injected into a branch of the portal vein failed to induce the slightest symptoms, testifying to the complete annihilation of the anthrax bacilli by the liver.

TESTS

The tests for liver function are as numerous as the advertised drugs having curative properties in diseases such as tuberculosis, where there are no known specific remedies. These tests run the gamut of the various liver functions and then run on into the purely arbitrary tests for dye excretion. Various dyes have been used, but the one most persistently experimented with in this country is the dye phenoltetrachlorphthalein, and

the history of its application to liver function testing carries a certain degree of interest.

In 1909, Abel and Rowntree determined by pharmacologic studies that phenoltetrachlorphthalein was excreted almost entirely by the liver and when injected intravenously is non-toxic. Rowntree, Hurwitz and Bloomfield and Whipple, Mason and Pughthal, in 1913, in the Bulletin of John Hopkins Hospital have articles showing experimental work with this dye wherein following injections of the dye the stools were collected for 24 to 48 hours and the amount of dye carefully estimated and they showed that the phenoltetrachlorphthalein output paralleled the amount of liver injury. In 1914, Krumbhaar, in summarizing the status of liver function tests, conceded that although no satisfactory single test for the final capacity of the liver has yet been accepted, the tetrachlor test of Rowntree promises the greatest value. In 1916, McNeil introduced a distinct improvement in the test by inserting the duodenal tube into the duodenum before the dye injection and, in 1921, Aaron, Beck and Schneider modified the technic and introduced a stable preparation of the dye and estimated the phenoltetrachlorphthalein elimination in the bile. They established the drip method of collection of bile from the duodenum by siphonage with the duodenal tube by the administration of 500 c.c. of water by mouth. They stressed the importance of the time of maximum color appearance and they considered the quantitative estimation of the dye excretion of little value. In 1922, P. F. Williams showed fairly constant end reactions in normal pregnant women and variable results in pathologic cases.

V. R. Deakin and E. A. Graham, Washington University, St. Louis, in an article in March, 1923, give an extensive bibliography and a summary of functional liver testing wherein they conclude that phenoltetrachlorphthalein is not a satisfactory substance to use in an hepatic functional test based on the quantitative estimation of its output in the bile; that there are too many factors which tend to prevent a complete collection of liver bile by the duodenal tube to warrant its use in such a procedure. In four dogs in which the dye was injected intravenously it was recovered in the duodenum in from 10 to 15 minutes after injection. They say:

"If the recent prominence which has been given the subject of non-surgical biliary tract drainage in medical literature by Lyon, Smithies, Einhorn, and others has done nothing else, it has served to emphasize the ease with which duodenal intubation may be accomplished, and this in turn has stimulated a revival of interest in what is still generally considered to be a closed chapter in the history of the liver and duodenum, the question of an hepatic functional test."

Working along similar lines, Piersol and Bockos, University of Pennsylvania, draw conclusions which are more favorable to the value of the test. They found in a series of fifty cases that, in a general way, a delay in the appearance time of the dye was proportionate to a decrease in the output and conclude that the estimation of the dye output in two hours is of more importance than recording the appearance time, although both should be considered. They found that in grossly pathologic livers the appearance time of the maximum color was twice as long as that in normal cases (23.2 minutes, as compared to 11.6 minutes), and the dye output averaged but one-eighth of the output in normal cases (2.71 mgm., as contrasted with 22.4 mgm.).

In 1922, S. M. Rosenthal did some original work and developed an improved phenoltetrachlorphthalein test for liver functioning in which he attempted to measure not what normal and pathologic livers would excrete, but how much they would not excrete; in other words, how much was retained in the blood plasma. He injected 5 mgm. per kilo of body weight and found that this dosage was removed from the blood stream very rapidly; where livers were normal, from 2 to 6 percent was found present in the blood plasma 15 minutes after injection and practically com-